QUARTERLY REVIEW of PEDIATRICS

Hypocalcemia and Tetany in the Newborn

Vol. 5 No. 4 November 1950

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Thanks for telling us...

you wanted a starting cereal made with RICE

NOW—GERBER'S RICE CEREAL IS READY

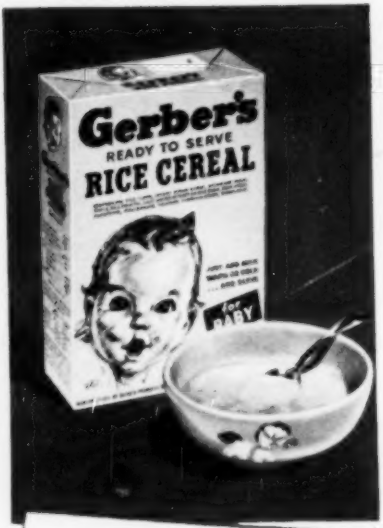
We're glad you told us that another one-grain cereal was needed to give some babies a better start and to increase the cereal variety for others all through babyhood. Helping you further sound infant nutrition is our business . . . our only business. So, after following the recommendations of leading bio-chemists, nutritionists, allergists and pediatricians—the new Rice Cereal is ready.

IT'S HYPO-ALLERGENIC. Gerber's new Rice Cereal is specially helpful with infants who may present wheat or oat allergy problems. Its B-vitamin supplementation is from rice bran extract plus crystalline thiamine, riboflavin and niacinamide.

IT WIDENS YOUR CHOICE of *one-grain* cereals. Now you are no longer limited to Gerber's Barley Cereal or Strained Oatmeal.

IT'S PARTICULARLY PALATABLE and has the Perfected-Texture babies like, too.

IT INCREASES THE VARIETY a mother can offer her infant or toddler. Now the Gerber Baby appears on four, dry, pre-cooked cereals.



Now! GERBER'S CEREAL "QUADS" in miniature sample boxes

The new Rice Cereal as well as Cereal Food, Strained Oatmeal and Barley Cereal! Order these new samples for your young mother clientele. Please write on your letterhead to Dept. RP 11-9 Fremont, Michigan.

Babies are our business
. . . our only business!

Gerber's
BABY FOODS

CEREALS • STRAINED FOODS • JUNIOR FOODS • MEATS

New!

FER-IN-SOL

For the prevention and treatment of iron deficiency anemia

FER-IN-SOL is a concentrated solution of ferrous sulfate, to be used in drop dosage for prevention and treatment of iron deficiency anemia.

Ferrous sulfate in an acidulous vehicle is widely accepted as the most effective form of iron for administration to persons of all ages.

Because of its pleasant citrous flavor, Fer-In-Sol is taken willingly by infants and children. It blends perfectly with citrus fruit juices and leaves minimum aftertaste.

The Fer-In-Sol dropper is conveniently calibrated for doses of 0.3 and 0.6 cc. (7.5 mg. and 15 mg. of iron). Only 0.3 cc. is required to provide the Recommended Daily Allowance of iron for infants and young children; 0.6 cc. provides the Recommended Daily Allowance for adults, including pregnant women.

Available in 15 and 50 cc. bottles with appropriately calibrated dropper.

MEAD'S

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U. S. A.

When training starts...

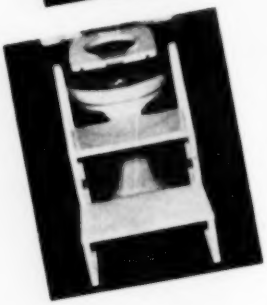
LITTLE TOIDEY

Whether you recommend baby be "conditioned" through the pleasant Little Toidey, before muscle control, or

you prefer the start be made after the toddler "knows what it's all about"

Toidey Habit Training Units can be helpful.

The recently developed Toddler's Toidey is particularly important for the child who starts second half of second year. It is also helpful if there has been a break in training and a new start has to be made. This Toddler's Toidey is all aluminum except that it has four suction cups on the underside of the seat so that this seat itself can be used on the toilet without the legs or the pan. This could be used as an emergency when traveling or later, when the child is two or three years old, in combination with Toidey Two Steps.



The Toddler's Toidey is also a Base for Little Toidey. The top is aluminum formed into a comfortable seat and enameled.

SPECIAL COURTESY DISCOUNT TO PHYSICIANS

The complete Toidey Habit Training Unit consists of Little Toidey (in plastic or wood), Toidey Base, Toideyette (deflector) and Specimen Collector. The complete unit or individual items are now available to members of the medical profession for their home or office use at a special courtesy discount of 20% from regular retail prices. Courtesy discount only on orders mailed directly to us.

THE TOIDEY COMPANY

Gertrude A. Muller, Inc.

FORT WAYNE, INDIANA, U. S. A.

Quarterly Review of PEDIATRICS

Vol. 5 No. 4 November 1950

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FOREWORD

The prime function of the QUARTERLY REVIEW OF PEDIATRICS is to make a survey, with a critical eye, of the important new contributions in every branch of pediatrics. Original reports are abstracted by pediatricians familiar with the subjects under consideration. The Editors and Editorial Board check over all abstracts and add interpretative or critical comments whenever necessary. The "Bookshelf" department reports informatively on new books the pediatrician should know about. Thus, within the covers of a single journal, there is brought together a concise chronicle of pediatric progress, well organized, reliable and complete. By exploring the entire expanse of medical literature the QUARTERLY REVIEW OF PEDIATRICS keeps its readers abreast of the most recent progress in all of pediatrics. A subscription to THE QUARTERLY REVIEW OF PEDIATRICS represents a continuous seminar on advances in pediatrics.

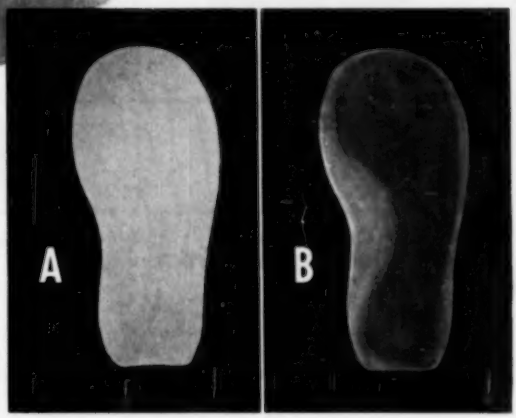
For convenience of reference the abstracts are grouped as follows:

1. Allergy
2. Anomalies, Genetics
3. Blood, Hemopoietic System
4. Cardiovascular System
5. Chemotherapy, Drugs, Poisons, Physical Agents
6. Clinical Pathology
7. Endocrine System
8. Eye, Ear, Nose and Throat
9. Gastrointestinal System
10. Genitourinary System
11. Growth, Puberty, Adolescence
12. History, Biography, Antiquities
13. Infectious Diseases, Acute
14. Infectious Diseases, Chronic
15. Liver, Kidneys and Spleen
16. Metabolic and Systemic Disorders
17. Milk; Infant and Child Feeding
18. Miscellaneous
19. Musculoskeletal System
20. Nervous System
21. Newborn Period, Prematurity
22. Nutrition
23. Parasitic Diseases
24. Pathology, Anatomy, Bacteriology
25. Physiology, Biochemistry
26. Psychology, Psychiatry
27. Public Health, Epidemiology
28. Respiratory System
29. Skin, Teeth, Hair
30. Social, Economic and Organizational Problems
31. Surgery, Anesthesia
32. Tumors

The Pediatric Bookshelf
New Books, Pamphlets
Announcements

Issues of the QUARTERLY REVIEW OF PEDIATRICS appear in February, May, August, and November. A cumulative index for each volume is included in the November number. Suggestions, correspondence and editorial communications should be addressed to Irving J. Wolman, M.D., *Editor*, The Children's Hospital, 1740 Bainbridge Street, Philadelphia 46, Pa. Subscriptions should be mailed to the Washington Institute of Medicine, 1708 Massachusetts Avenue, N.W., Washington 6, D. C. Annual Subscription: $11.00. Three years: $28.00.

THE PROOF IS IN THE FOOTPRINT



A Here's a brand-new Jumping-Jack child's shoe. Note the unique, patented "all-in-one" sole and heel!

B Same shoe, after 6 months' wear. Arch shows almost no wear, proving child is forming own natural arch!

JUMPING-JACKS foster healthy foot development!

Jumping-Jacks provide the necessary gentle, firm support for active young feet . . . without artificial restraint from stiff counters or shanks. The one-piece sole and heel encourages healthy muscle growth. The proof's in the footprint . . . *natural, healthy development* with Jumping-Jacks!

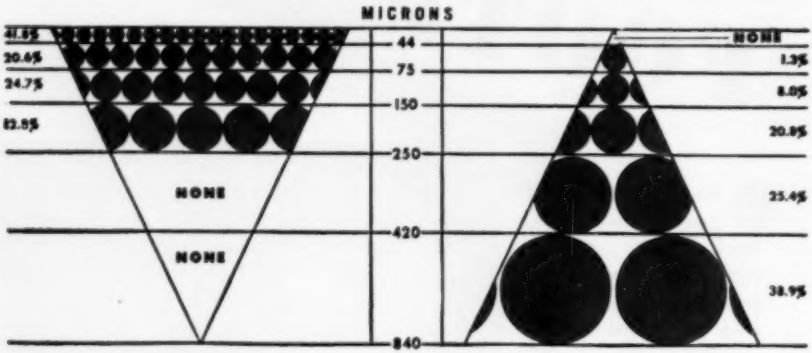
JUMPING-JACKS®
FLEXIBLE SHOES FOR HARD WEAR

VAISEY-BRISTOL SHOE COMPANY, INC. • ROCHESTER 3, NEW YORK

MONETT, MISSOURI • SKOWHEGAN, MAINE • MARIONVILLE, MISSOURI
MADE IN CANADA BY THE SAVAGE SHOE COMPANY, LTD., PRESTON, ONT.

Particle Size

AN INDEX TO READY DIGESTIBILITY



Libby's STRAINED and HOMOGENIZED CARROTS

CARROTS MERELY STRAINED

THE ready digestibility of Libby's Strained AND Homogenized Baby Foods, and their early tolerability, are graphically shown as physical changes which Libby's exclusive process of homogenizing brings about.

For instance, in carrots that have only been *strained*, less than 30% of the food substance presents particles under 250 microns in size—more than 70% is composed of particles up to and over 840 microns in size. BUT when this substance undergoes Libby's *homogenizing* process, there remain no particles over 250 microns in size; 87% are smaller than 150 microns.

Thus digestion is facilitated, and utilization of contained nutrients, such as iron, is enhanced. Since cellulose fibers are comminuted to ultra-small size, Libby's Homogenized Baby Foods may be fed with safety as early as the fifth week of life and are well tolerated.* Yet this feature carries no price penalty, for Libby's cost the mother no more than ordinary, merely strained, baby foods.

**Reprints of clinical studies are available on request.*

Libby, McNeill & Libby • Chicago 9, Illinois

Libby's

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

HOMOGENIZED BABY FOODS

Drop-cillin

50,000 UNITS* in a dropperful

9 cc. bottle contains 600,000 units.

Dram-cillin

100,000 UNITS* in a teaspoonful

60 cc. bottle contains 1,200,000 units.

Both with a delicious vanilla flavor.

*buffered penicillin G potassium

White Laboratories, Inc.,
Pharmaceutical Manufacturers, Newark 7, N. J.

eating milk is fun!

delicious and nutritious, too

The simple expedient of altering the form and texture of liquid milk—easily and quickly accomplished with rennet—frequently solves a number of eating problems. • Children—and oldsters, too—who (for one reason or another) tire of *drinking* milk, enjoy *eating* delicious rennet-custards. Quickly prepared, and in no way changing the nutritive values of uncooked milk, rennet-custards may be used as desserts or as between-meal snacks—varied in many ways through the availability of six different flavors and innumerable toppings. • Remember rennet next time you have a milk problem!

"JUNKET" is the trade-mark of Chr. Hansen's Laboratory, Inc. for its rennet and other food products and is registered in U.S. Patent Office.

"JUNKET" BRAND FOODS
DIVISION
Chr. Hansen's Laboratory, Inc. • **LITTLE FALLS, N. Y.**

Key H-113

"Junket" Rennet Powder
—sweetened, six flavors.

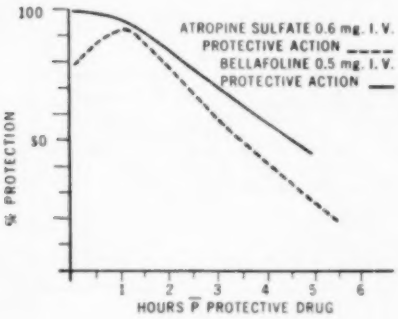
"Junket" Rennet Tablets
—unsweetened, unflavored (particularly for very young infants and diabetics).

New Form of Levorotatory Belladonna Alkaloids for Selective Spasmolytic Action

The practical value of an antispasmodic depends upon its degree of desirable spasmolytic effect, convenience of administration and patient acceptability.

Degree of spasmolytic effect of belladonna alkaloids rests upon the intensity of parasympathetic inhibition. Pure levorotatory belladonna alkaloids (Bellafoline) are more potent and selective than belladonna alkaloid mixtures in producing this spasmolytic effect, at the same time minimizing the undesirable cerebrospinal effects.

Studies by *Kramer and Ingelfinger*, (M. Clin. North Amer., **Boston No.**: 1227, (1948) demonstrate the highly efficient action of Bellafoline. By balloon-kymograph studies on the human intestine they found that most commonly used antispasmodics are less effective than atropine (standard dose: 1/100 gr.). Bellafoline was the outstanding exception. It surpassed atropine in both degree and duration of action.



Protecting capacity of Anticholinergic Agents against bronchospastic effects of I. V. Mecholyl. From graphs in Beakey, et al. * Annals of Allergy, Vol. 7, No. 1. Jan.-Feb. 1949

The antispasmodic effect of Bellafoline is augmented by the addition of a small dose of phenobarbital thereby reducing excitability and tension.

Such an association of Bellafoline and phenobarbital is *now available in the form of Elixir Belladenal* and may therefor be valuable for treatment of spastic disorders that are associated with hyperirritability and anxiety.

The several requirements mentioned for a practical, spasmolytic agent are fulfilled by *Elixir Belladenal*. In addition to high efficacy, it is specifically designed for patient acceptability and convenience of dosage regulation in the full range of antispasmodic application. It is especially serviceable in pediatrics and in those adults where the use of tablets and capsules is impractical. The teaspoonful dose contains Bellafoline (levorotatory alkaloids of belladonna leaf) 0.0625 mg. and phenobarbital 12.5 mg.

ELIXIR BELLADENAL is indicated in spastic disorders such as: feeding problems in infants and children, spastic colitis, cardiospasm, pylorspasm, peptic ulcer, pseudo-ulcer syndrome, biliary colic, renal colic and dysmenorrhea.

Also available in tablet and suppository forms. Professional Samples and Literature are available upon request.

Sandoz Pharmaceuticals

DIVISION OF SANDOZ CHEMICAL WORKS, INC.
68 CHARLTON STREET, NEW YORK 14, NEW YORK

The only broad-spectrum antibiotic available as an elixir

TERRA

Each teaspoonful (5 cc.) provides 250 mg. of *Terramycin Hydrochloride*

plus

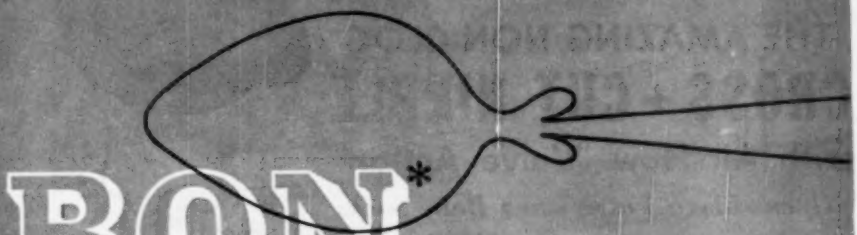
cherry-color appeal

plus

cherry-mint flavor

Antibiotic Division **Pfizer**

High dosage concentration ensures therapeutic efficacy without requiring new and unwieldy dosage schedules.



BON*

Brand of

TERRAMYCIN ELIXIR

Suggested for:

pneumonia (pneumococcal, mixed bacterial, Friedländer's, primary atypical); respiratory infections including bronchitis, pharyngitis, laryngotracheitis, tracheobronchitis, sinusitis, tonsillitis, otitis media, bronchiectasis, pulmonary infections associated with pancreatic insufficiency, pertussis, influenza; diphtheria (adjuvant therapy); urinary tract infections due to Terramycin-sensitive organisms including E. coli, A. aerogenes, Staph. aureus, etc.; surgical conditions including soft tissue lesions; ophthalmic infections including catarrhal conjunctivitis; pneumococcal infections; streptococcal infections; meningococcal infections; brucellosis (abortus, melitensis, suis); acute infections due to E. coli and A. aerogenes; shigella infections; salmonella infections; hemophilus infections; tularemia; plague; anthrax; gas gangrene due to Cl. welchii, E. coli, and nonhemolytic streptococci; cellulitis and abscess due to Actinomyces; Bacteroides bacteremia; spirochetal infections; rickettsial infections; viral and viral-like infections; gonococcal urethritis; amebiasis.

Available at prescription pharmacies in bottles containing 1 fl. oz.

*Trade Mark

CHAS. PFIZER & CO., INC., *Brooklyn 6, N. Y.*

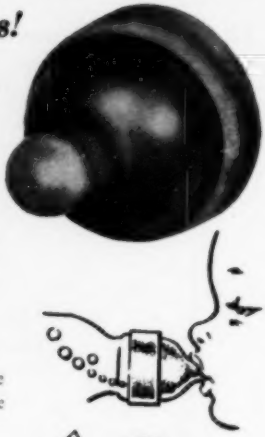
NO MORE CLOGGED NIPPLES!

New Advance In Feeding Infants!

Baby-All Introduces

THE AMAZING NON-CLOG
CROSS + CUT NIPPLE
with Air-Flow "Valve Action"

Opens like a Valve when Baby Sucks



The problem of nipples clogging is now overcome with the new Baby-All Cross + Cut Nipple that opens like a valve when baby sucks. No holes to clean. It allows milk to flow as fast as baby demands. Hospital tested. Used with the Baby-All Shielded Nurser—*ideal for Terminal or Aseptic Sterilization.*

For Terminal Sterilization

Baby-All FORMULA & STERILIZER OUTFITS

Terminal Sterilization is the latest technique approved by American Hospital Association and American Academy of Pediatrics. With Baby-All sterilizers, mothers at home can now sterilize baby's formula together with bottles, nipples, and shields *all in one operation.* Can also be used for the Aseptic Method. Formula and nipple remain *untouched and sterile to moment of feeding.*

A Simple Method Every Mother Can Follow

Electric and Non-Electric Models.
Accommodate all standard nursers.

Demonstrated to Mothers in Hospitals and Maternity Centers. Sold in Surgical Stores, Infants Departments and Drug Stores.

FREE Write for your copy of paper "Terminal Sterilization of Infant Formula in the Home" and for literature on the new automatic all night Vaporizer.

SANIT-ALL PRODUCTS CORP.

Greenwich, Ohio

Why so many Doctors Keep Babies on Carnation the Entire 1st year

1.

During Formula Days

Carnation Evaporated Milk, with water and carbohydrates, is the safe and sure formula that can be adjusted to fit each baby's individual requirements.

2.

When baby goes off Formula

Carnation diluted with an equal amount of water is nourishing whole milk in its most digestible form.

3.

From bottle to Cup Drinking

Carnation's familiar flavor encourages baby's acceptance of cup-drinking . . . its uniformity discourages digestive upsets that may be caused by radical changes.

Doctors agree that radical changes in the care and feeding of a baby should be avoided—*especially* during the important first year of its life. That's why so many doctors advise mothers against taking baby off formula too soon . . . or changing to a different form of milk when formula days are over.

For the constant uniformity of Carnation Evaporated Milk . . . in butterfat, milk solid content, curd tension and viscosity . . . are positive factors in eliminating digestive upsets throughout baby's first year. And because Carnation is not only pasteurized, but also sterilized *after* the can is sealed, it is doubly *safe* milk.

Every can of Carnation Milk comes from cows checked by Carnation's *own* inspectors . . . is tested in Carnation's *own* receiving stations . . . is processed under rigid control in Carnation's *own* evaporating plants. It's a brand you can recommend *by name*, as doctors have been doing for more than 50 years . . . with complete confidence that there is no better, safer milk for babies.

8 out of 10 mothers raising their babies on Carnation report that it was recommended by their doctor

The Milk Every Doctor Knows

"From Contented Cows"

"Upper Respiratory"—

aspergum® for Relief

For childhood colds, sore throats and other febrile or painful conditions, Aspergum provides acetylsalicylic acid in easy-to-take, deliciously flavored, *chewing gum form.*

The antifebrile, analgesic medication is quickly absorbed, reaches the blood stream rapidly, producing a mild general analgesia and antipyresis in children.

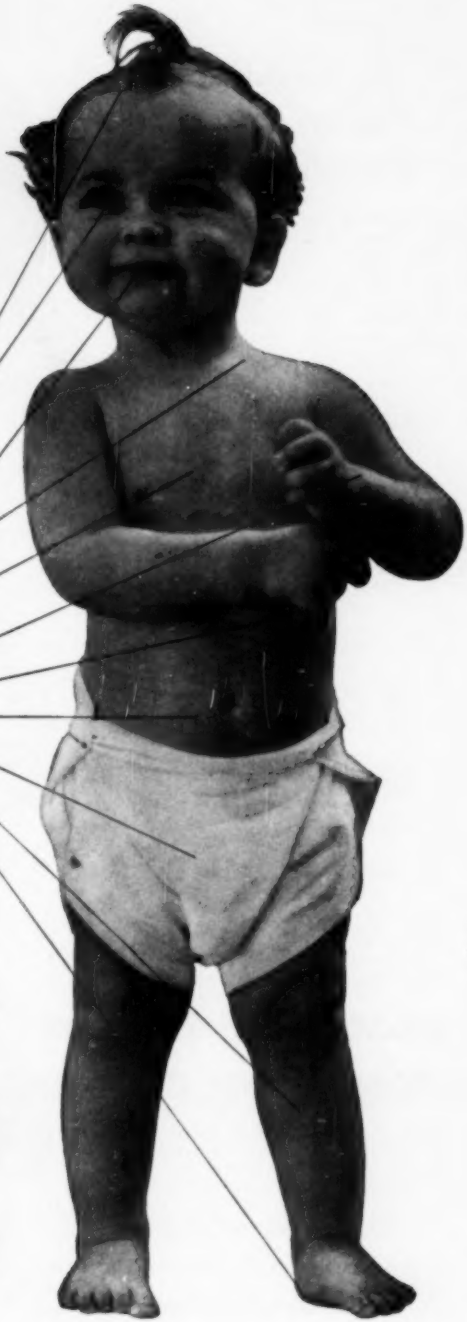
Ideal for post-tonsillectomy cases, Aspergum offers immediate local relief of pain, encourages an earlier return to a full diet—convalescence is hastened.

Just 3½ grains of acetylsalicylic acid per tablet —a dosage and form uniquely fitted to childhood requirements. Promoted ethically.

WHITE LABORATORIES, INC.,
Pharmaceutical Manufacturers, Newark 7, N. J.

from *head* to *toe*

CEREVIM-fed children showed greater clinical improvement, in the following nutrition-influenced categories, than children fed on ordinary unfortified cereal or no cereal at all:[1]



Here's why: **CEREVIM is not just a cereal.**

Much more: **CEREVIM provides *8 natural foods:* whole wheat meal, oatmeal, milk protein, wheat germ, corn meal, barley, Brewers' dried yeast and malt – PLUS added *vitamins* and *minerals*.**

CEREVIM

CEREALS + VITAMINS + MINERALS

1. "A Study of Enriched Cereal in Child Feeding" Urbach, C.; Mack, P. B., and Stokes, Jr., J. Pediatrics 1:70, 1948.

*Cerevim contains neither vitamin A nor C, but apparently exercises an A-and-C sparing effect attributed to its high content of predigested protein and major B vitamins.

SIMILAC DIVISION M & R DIETETIC LABORATORIES, *Columbus 16, Ohio*

White's Multi-vi drops

WATER-MISCIBLE • NON-ALCOHOLIC

In one inexpensive drop-dosage preparation — all essential vitamins for prevention and treatment of multiple vitamin deficiencies in infants and children —

Formula: Each 0.6 cc. contains:

Vitamin A 5000 U.S.P. units • Vitamin D_2 1000 U.S.P. units
Thiamine Hydrochloride 1.0 milligram
Riboflavin 0.4 milligram
Pyridoxine Hydrochloride 1.0 milligram
Panthenol* 2.0 milligrams • Niacinamide 10.0 milligrams
Ascorbic Acid 50.0 milligrams
*a specially-prepared, more stable analogue of pantothenic acid.

White Laboratories, Inc., Pharmaceutical Manufacturers, Newark 7, N. J.

How times (to start meat) have changed!

Where meat was once withheld until seven or eight months, today it takes its rightful place beside baby's first solid foods. In a recent survey among mothers who are feeding meat, 47.4% stated that doctors recommended it for their babies at *four months* or earlier. Some doctors advocate 2 weeks.

Doctors, and mothers too, have been quick to welcome the addition of meat to baby's diet early in life. It was only four years ago that Swift first developed specially prepared meats, and inaugurated clinical studies to evaluate meats' specific benefits. Since then, babies have been started on meat at younger and younger ages.

Ounce for ounce, no other infant food supplies more of the complete proteins, B vitamins and natural food iron that baby needs than Swift's Meats for Babies. Laboratory-controlled preparation insures maximum retention of nutrients. Swift's Meats for Babies and Juniors are ready to serve at a saving of almost half the cost of home-prepared meats.

All nutritional statements made in this advertisement are accepted by the Council on Foods and Nutrition of the American Medical Association.

Clinical studies show benefits of early meat-feeding

TO PREMATURES: Nutrients in Swift's Meats for Babies well tolerated, well utilized at this age. Also, significant retention of iron: *University of Rochester.*

AT SIX WEEKS: Increasing protein intake 25% by addition of Swift's Meats for Babies to formula, promoted hemoglobin and red blood cell formation: *Leverton and Clark, "Meat in the Diet of Young Infants," J. A. M. A. 134, (August) 1947.*

WITH ALLERGY CASES: A formula using Swift's Meats for Babies offers an effective milk substitute for infants allergic to milk proteins: *"Nutritive Value of Mineral-Enriched Meat and Milk," McQuarrie & Ziegler, Pediatrics, Vol. 5, No. 2, (February) 1950.*

SWIFT... *foremost name in meats*

... *first to develop and clinically test 100% Meats for Babies*

Purity

you can see

in Crystal Clear

Immune Serum Globulin · CUTTER

You have *visible evidence* of purity in a vial of Immune Serum Globulin-Cutter—compare it as a product of choice for measles' modification and prevention.

Crystal Clear—Look at the highly purified homologous protein in the Cutter vial. *See* the crystal clear, hemolysis-free gamma globulin.

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Quarterly Review
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EDITORIAL

Hypocalcemia and Tetany in the Newborn.

The growing recognition that hypocalcemia with or without tetany is frequent in the newborn infant is responsible for a number of recent important studies on the physiology of calcium in the neonatal period. Attempts to evaluate the complex factors involved have been impeded, unfortunately, by the absence of a uniform concept of the clinical disturbance under investigation. A fundamental distinction that is essential to the fruitful solution of this problem was made in the excellent review by Katherine Dodd (Hypocalcemic States, Pediatrics 2: 737, 1948)—"In general, hypocalcemic states are manifested by signs and symptoms of tetany." The term neonatal tetany should thus be limited to its descriptive meaning. Convulsive disturbances are not uncommon in the neonatal period and may bear no relationship to hypocalcemic tetany. Hypocalcemia with or without a disturbed phosphorus level in the blood seems to be the physiologic incitant of the symptoms as a rule, but the causative mechanisms which interact to produce this imbalance are not yet clearly elucidated.

Symptoms of tetany seem more frequent in newborn infants started on cow's milk mixtures than in those nursed by the mother. One major influence seems to be the much higher content of phosphorus in cow's milk and its lower Ca : P ratio as compared to breast milk. One obvious approach to prevention of this metabolic disturbance, therefore, would be a set of experiments designed to determine the most beneficial range and ratio of Ca : P in cow's milk formulas.

Normal Levels of Calcium and Phosphorus in the Newborn Infant. For years it has been known that during the first few days of life the serum calcium level falls transitorily and the serum phosphorus seems

to rise. These changes are well exemplified in the figures of Gittleman and Pincus who recently summarized an extensive study of the chemical composition of the blood in artificially-fed newborn infants in Brooklyn, N. Y. (Table I).

Table I—*Frequency Distribution of Serum Calcium Levels (data of Gittleman and Pincus)*

	Serum Calcium (mg. per 100 ml.)			
Age	9 to 11	8 to 9	7 to 8	< 7
2–24 hr.	83.3%	16.7%	1.4%	
24–36 hr.	77.1%	17.1%	5.7%	
48 hr.	60 %	33 %		6.6%
3–15 days	52.6%	23.7%	23.7%	

For the first 24 hours these infants had received feedings of glucose water only. They were then given an evaporated milk and water mixture in the proportion of 1 to 2, with 5% added dextri-maltose. The serum phosphorus values in these same infants had a tendency in the reverse direction. In the first 24 hours more than 90% of those tested had serum levels less than 7 mg. per 100 ml., whereas after three days nearly 80% had levels higher than 7.4 mg. per 100 ml. (Table II).

Table II—*Frequency Distribution of Serum Phosphorus Levels (data of Gittleman and Pincus)*

	Serum Phosphorus (mg. per 100 ml.)					
Age	2–3.8	4.1–4.9	5–6.9	7–7.8	7.1–10.1	7.4–12.3
2–24 hr.	5.4%	17.5%	68.9%	8.1%		
24–36 hr.	5.7	14.3	65.7	14.2		
48 hr.		5.0	50.0		45%	
3–15 days		21.6%				78.3%

The serum potassium ranged between 17 and 18 mg. per 100 ml. The protein concentration varied between 4.6 and 6 Gm. The phosphatase concentration varied between 2.6 and 13.6 units (Bodansky).

Tetany in the Newborn. A careful study of the changes in serum calcium and phosphorus as related to tetany in the neonatal period is that of Dodd and Rapoport. Blood analyses were carried out on 24 fullterm infants in a newborn nursery in Cincinnati, Ohio. These infants exhibited clinical signs broadly suggestive of mild tetany, such as disturbances in breathing or feeding, vomiting, edema, drowsiness, stiffness, irritability, convulsions, crowing, Chvostek's sign, or ankle clonus. As controls, 48 other fullterm infants free from such symptoms were similarly studied. Parallel investigations were also carried out on two series of fullterm newborn infants with manifest tetany, and one series of premature infants in whom the signs of tetany were merely

suggestive. Striking correlation of the serum calcium levels to these clinical disturbances is shown in Table III.

Table III—*Distribution of values for serum calcium in newborn infants with suggestive or manifest tetany (Dodd and Rapoport)*

No. of Infants	State of Maturity	Symptoms of Tetany	Serum Calcium (mg. per 100 ml.)				
			> 10	10 to 9	9 to 8	< 8	< 7
48	Fullterm	None	23	20	4	1	
24	Fullterm	Mild to Moderate	3	5	13	3	
9	Fullterm	Prominent	0	0	4	4	1
33	Fullterm and Premature	Prominent	0	0	8	(7 to 8) 16	9
12	Premature	Mild to Prominent	0	0	5	(7 to 8) 4	3

The infants in this series who were asymptomatic usually had serum calcium values above 9 mg. per 100 ml. Among infants with mild tetany, in contrast, low calcium values were frequent but by no means universal. The severity of symptoms and the depression of calcium levels seemed to parallel each other, although there were a few infants with prominent symptoms whose calcium levels fell in the higher range of 9 to 8 mg. Since none of the infants with manifest tetany and only a few of those with suggestive symptoms had values above 9 mg. per 100 ml., a serum level above 9 mg. in a newborn infant makes the diagnosis of tetany unlikely.

The symptoms attributed to tetany made their appearance between the first and thirteenth day of life. Most of the infants seem to have been born without difficulty and were of normal weight at birth. Males predominated. The range of clinical manifestations was wide, with vomiting, convulsions, cyanosis, hemorrhages and edema (in 5 instances scleroderma) as the severest symptoms. The symptoms at times suggested cerebral birth injury or intestinal obstruction. The serum phos-

Table IV—*Distribution of values for serum phosphorus in newborn infants with suggestive or manifest tetany (Dodd and Rapoport)*

No. of Infants	State of Maturity	Symptoms of Tetany	Serum Phosphorus (mg. per 100 ml.)		
			< 6	6 to 8	> 8
47	Fullterm	None	23	22	2
16	Fullterm	Mild to Moderate	10	4	6
8	Fullterm	Prominent	2	2	4
25	Fullterm and Premature	Prominent	7	8	10
11	Premature	Mild to Prominent	5	6	0

phorus levels in these same series of infants are reproduced in Table IV. It was concluded that levels above 8 mg. are extremely uncommon in normal newborn infants but occur in about one-third of the infants with clinical tetany. There was a suggestive trend to values above 8 mg. with increasing severity of symptoms, except in the premature infants in whom high phosphorus values were not seen. The level of nonprotein nitrogen was often elevated in the infants with severe manifestations.

In Dodd and Rapoport's series the serum alkaline phosphatase was normal in all except one infant who had hepatic damage from erythroblastosis fetalis. There were less than 10 Bodansky units in 12 of 14 infants tested; in one of the others it fell between 12 and 20 units (high normal range). This is in striking contrast to rachitic tetany, in which the phosphatase is elevated with a fair degree of consistency.

Known Factors Contributing to Hypocalcemia of the Newborn. A number of influences seem able to affect the levels of serum calcium and phosphorus during the newborn period. Whether or not tetany will become manifest depends to some extent upon these influences, although much remains to be learned about each of them.

Illnesses affecting the acid-base equilibria such as vomiting with hypochloremic alkalosis, hyperventilation with respiratory alkalosis, postacidotic imbalances, and toxemia from acute infections can induce tetany at any age, and are occasionally operative during the newborn period. *Cerebral hemorrhage, erythroblastosis fetalis, prematurity,* and *maternal diabetes or pre-diabetes* are sometimes associated with hypocalcemia and tetany.

The *intake of calcium and phosphorus* in the diet and the ratio between these substances may play a significant role in those infants with tetany who are old enough to receive feedings. Bakwin was the first to demonstrate that administration of phosphate solutions to newborn infants encouraged the tendency to hypocalcemia, and suggested that the high content of phosphorus in cow's milk may be of causative significance in neonatal tetany. Gardner, MacLachlan, Pick, Terry and Butler have re-emphasized this difference. Cow's milk has a lower Ca : P ratio and higher total phosphorus concentration than human milk. These differences are accentuated in some of the proprietary milk preparations designed for infant feeding, as a consequence of phosphate-containing "stabilizers" added during manufacture. The analyses of Gardner et al showed the range in Ca : P ratio for representative samples of human milk in the first few days of lactation to be from 1.9 to 2.7. That for fresh cow's milk was 1.27, for evaporated milk, 1.05, and for spray dried cow's milk, 1.00. In the human milk samples the phosphorus varied from 8 to 15 mg. per 100 ml., whereas in cow's milk feeding mixtures it was 50 to 98 mg. In 2 newborn infants fed breast milk the serum phosphorus levels remained uniformly below 6 mg. per 100 ml. over the first 10 days of life. In 4 others fed diluted evaporated milk the values were higher.

The total serum calcium was around 8.8 and 8.6 in the breast-fed infants; it was 6.2 and 6.1 in the bottle-fed infants. The blood of the bottle-fed infants was higher in nonprotein nitrogen, probably because of the greater protein intake. Metabolic balance studies with one infant receiving consecutive modifications of cow's milk showed that the changes in serum calcium and phosphorus were minimized by increasing the Ca : P ratio of cow's milk to that of human milk or by reducing the total mineral content by liberal dilution with water.

The *renal excretion of phosphate* in the neonatal period seems to be dependent upon the amount of phosphate filtered through the glomeruli and upon the efficiency of tubular reabsorption. McCrory, Forman, McNamara and Barnett have demonstrated that infants who have or have had elevated endogenous levels of serum phosphate (with decreased levels of serum calcium) display a diminished capacity for renal excretion of phosphate. The decreased renal excretion of phosphate could result from either decreased filtration or increased reabsorption; both mechanisms may be involved.

Hypoparathyroidism also appears to contribute to the pathogenesis of neonatal tetany. Removal of the parathyroids from a human being brings about a decreased excretion of phosphorus in the urine, followed by a rise in serum phosphorus, a fall in serum calcium, and a diminished output of urinary calcium (Albright and Reifenstein). With parathyroid stimulation this sequence of events takes place in the reverse direction. There are indications from animal experiments that the parathyroids hypertrophy when the dietary Ca : P ratio is low. The feeding of cow's milk mixtures without mineral modification may call for a higher level of activity by the parathyroids than with breast milk. There may be a lag interval before this occurs. Suggestive confirmation lies in the observation of Kaplan, and Gardner and associates, that the parathyroids of 8 autopsied newborn infants fed cow's milk had "idiopathic hypertrophy," whereas those from 7 breast-fed newborn infants were "normal." Proper evaluation of the contribution of parathyroid activity to the pathogenesis of neonatal hypocalcemic tetany warrants further investigation in view of the studies of McCrory, Forman, McNamara and Barnett on the renal excretion of phosphate by normal newborn infants.

Vitamin D deficiency seems not to be responsible for the hypocalcemia and tetany seen in such newborn infants, since clinical or roentgenologic signs of rickets are not ordinarily found and the serum alkaline phosphatase is not elevated. On the other hand, it must not be forgotten that histologic irregularities characteristic of rickets have been demonstrated by Ranström anr von Sydow in 20 of 69 Swedish infants (29%) dying during the first day of life. Likewise, Eliot and Park reported the existence of histologic rickets in 21% of 90 infant autopsies during the first month of life. In many such infants personally studied

these histologic changes are minimal and seem of brief duration, reflecting perhaps a disturbance in bone growth during the course of the terminal illness.

Comment. In spite of the limited knowledge concerning physicochemical disturbances leading to the manifestations of hypocalcemic tetany, it would seem desirable that the demonstration of hypocalcemia be a prerequisite to the diagnosis. The influences which depress the level of serum calcium should be clearly separated from the mechanisms operating to precipitate the signs and symptoms of tetany in existing hypocalcemic states. The hypocalcemic state must be qualified by the presence (or absence) of signs and symptoms—not defined by them. The concurrence of clinical tetany and hypocalcemia may suggest a causal relationship but this has not been clearly established. Some authors use the therapeutic response to calcium as a further criterion. The prompt alleviation of the symptoms and signs interpreted as manifestations of tetany by calcium therapy is empirically accepted as demonstrating an underlying hypocalcemia, even though the physiologic basis for this response is not as yet clear or even whether hypocalcemia is the cause of the symptoms. Newborn infants can experience marked distortions in serum electrolyte concentrations either as a result of primary diseases or in association with acidosis and dehydration resulting from feeding difficulties, birth injury, anoxia, deficient fluid intake, etc.

The full story of the interaction of the factors responsible for neonatal hypocalcemia is thus still far from complete. Once the mechanisms are elucidated an intelligent program of preventive therapy will be feasible.

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(NOTE: For abstracts of other recent papers devoted to disturbances in calcium and phosphorus metabolism in older infants and children, see section on "Physiology, Biochemistry" in this issue of the QUARTERLY REVIEW OF PEDIATRICS.)

1. Allergy

See Contents for Related Articles

2. Anomalies, Genetics

Study of the Etiological Factors in 304 Cases of Congenital Cardiac Malformations (*Enquête étiologique sur 304 malformations cardiaque congénitales*). *M. Lamy and O. Schweisguth, Paris, France.* Ann. pædiat. 174: 65–68, Jan.–Feb. 1950.

In 304 cases of congenital cardiac malformations of various types, a study of possible etiologic factors was made. There was consanguinity of the parents in only 10 cases (3.3%); cardiac malformations were found in the patient's family in 24 cases, and other types of malformation in the family in 20 cases. Direct heredity of a cardiac malformation was found in only 3 cases. In 10 cases, a cardiac abnormality was present in a brother or sister of the patient; in these cases the parents were normal and consanguinity was noted in only 1 instance. In other familial cases, a cardiac abnormality was present in collaterals. The other malformations, not involving the cardiovascular system, as shown in the family history, also occurred chiefly in collaterals. These findings suggest the possibility of a recessive mendelian heredity, but this theory was not supported by the finding that in 2 cases of identical twins, 1 twin showed the abnormality and the other did not.

A study of the patients themselves showed that 43 (14%) had other abnormalities not involving the cardiovascular system; of these mongolism was the most frequent (10 cases). A study of the mother's pregnancy was made in 225 cases. There were only 2 cases in which a history was obtained that showed that an eruption, which might have been rubella, occurred in the first two months of pregnancy; 1 of these 2 children was a deaf-mute, the other showed congenital cataracts in addition to the cardiac abnormality. Other types of infection had occurred in 6 cases; in 2 cases this was a throat infection, and in 1 of these cases the child showed mental deficiency and congenital cataracts in addition to the cardiac abnormality, but in none of these cases could the role of the infection in the production of the congenital abnormality be proved. In 22 other cases, some other abnormal condition had complicated the pregnancy; in 7 cases abnormal bleeding had occurred in the first three months of pregnancy and in some of these corpus luteum had been used in treatment. In some there was a history of poisoning by drugs, in others a history of an injury (including attempted abortion) in the early months of pregnancy. In none of these cases could the etiologic significance of these occurrences be determined. Further and more extensive studies are necessary to determine the cause of congenital cardiac malformations.

Gaucher's Disease: Cases in Five Related Negro Sibships. *C. Nash Herndon and John R. Bender, Department of Medical Genetics, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, N. C.* Am. J. Human Genetics 2: 49–60, March 1950.

Five instances of Gaucher's disease are described in a Negro kindred, the diagnosis being proved by autopsy in 4 cases. The pedigree of this family is presented, showing the 5 cases to be in 5 separate but closely related sibships. The parents of 4 of the affected children were second cousins. The genetic mechanism responsible for Gaucher's disease in this family can be deduced with reasonable reliability from the pedigree. Gaucher's disease appears to be due to the action of an autosomal recessive gene. In the homozygous state this gene gives rise to an intracellular metabolic defect which results in the deposit of kerasin in the reticulo-endothelial cells. Evidence presented in other family reports is compatible with this conclusion. 18 references. 3 figures.

The practical value of a genetic survey such as this would be much enhanced if the family group had been given diagnostic bone marrow examinations. Marrow aspirates will frequently reveal the existence of Gaucher's disorder in individuals who, on physical examination, seem free of the disease. Such individuals are not infrequent in families which contain active cases of Gaucher's disease. Such a survey would explore the possibility that the florid disease has a homozygous inheritance, whereas the subclinical form is of heterozygous origin—a possibility not explored or even considered by the authors of this paper. Should this prove to be true a formula might be adduced from the data which would permit an estimate of the occurrence of the heterozygous or "carrier" state within any ethnic group, provided the frequency of the florid or homozygous disturbance was known. Diagnosis and eugenics would both profit by such knowledge, since heterozygotes could then be advised against marrying each other.—Editor.

Acute Form of Gaucher's Disease in Three Siblings (*Acute vorm de ziekte van Gaucher by drie kinderen uit één gezin*). *L. Strengers, The Hague, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 237–49, 1949.

In a family of 7 siblings, the 3 younger ones all died at the age of about 10 months from the acute form of Gaucher's disease. They showed spleno-hepatomegaly, opisthotonus even in such a degree that the children were unable to lie on their backs, strabismus and a markedly pale color. The Gaucher cells in the marrow obtained from the tibia were not characteristic early in the disease, but became typical later on. In one of the children splenectomy was performed without benefit; the child died six months later. Roentgenograms of the lungs showed fan-like shadows at the hila. At autopsy Gaucher cells were found in the

lungs. Although during life distinct cerebral disturbances were present, at autopsy no cerebral lesions were found.

Neither the mother nor the other children showed any morbid symptoms, but the bone marrow could not be examined. The father who had no complaints was found to be suffering from myeloid leukemia. 11 references. 1 table.—*Ph. Arons.*

Gaucher's disease is familial. Affected members of a family may show clinical disturbances in varying degrees. A complete workup of any case should always include a survey of all parents and all siblings by bone marrow aspiration. Unfortunately no treatment is known except splenectomy, should that organ become too bulky.—EDITOR.

Hereditary Congenital Anomalies of the Skeleton (Dislocation of the Capitulum radii: Dislocation, Hypoplasia or Absence of the Patella) and of the Nails (*Erfelijke afwijkingen van skelet [luxatie van het capitulum radii; luxatie, hypoplasie of ontbreken van patella] en nagels*). *I. S. Wildervanck, Groningen, The Netherlands.* Nederl. tijdschr. v. geneesk. 93: 2576–83, July 30, 1949.

A description is given of a family of 53 members in five generations, 22 of whom show the following anomalies in different combinations:

Disturbances in the elbow-joint: dislocation backwards of the capitulum radii; hypoplasia of the capitulum humeri; exostosis of the processus coronarius ulnae. The processus styloidei radii is overdeveloped.

Disorders of the knee-joint: hypoplasia or absence of the patella; dislocation of the patella to the side, most likely caused by an underdevelopment of the musculus vastus medialis; malformation of the condyles and of the tuberosity of the tibia.

Malformations of the nails: absence or dystrophy of the nails, this is at worst in the nails of the thumbs, the least in the little fingers. It is the same as that of another anomaly, namely, lunulae, which are very great and triangular.

There may be flexion of the fingers, especially of the little fingers.

All these anomalies are irregularly dominant. They are probably caused by three incompletely linked genes. 14 references. 1 figure.—*H. I. J. M. Bartels.*

3. Blood, Hemopoietic System

Thrombocytopenic Purpura: The Prognostic and Therapeutic Value of the Eosinophil Index, an Analysis of 100 Cases. *Steven O. Schwartz and Sherman R. Kaplan, Cook County Hospital, Chicago, Ill.* Am. J. M. Sc. 219: 528–33, May 1950.

This is an investigation into the value of the Eosinophil Index (i.e., the number of marrow eosinophils per 1,000 granulocytes—both at the metamyelocytic stage or later) as a prognostic tool in thrombocytopenic

purpura hemorrhagica. One hundred patients with "primary" thrombocytopenic purpura were studied, aged 20 months to 90 years. Eleven were under 6 years of age, 7 between 6 and 10 years, and 23 between 11 and 20 years. Marked platelet reduction, delayed clot retraction, prolonged bleeding time, petechiae, ecchymoses, and increased capillary fragility characterized all cases.

In the normal marrow the number of eosinophils is said to be less than 50 per 1,000 maturing granulocytes (index less than 50). For this reason, and in light of earlier experiences, the authors have come to use 50 as the dividing point. An eosinophil index of more than 50 is said to be "high"; less than 50, "low."

On this basis the cases were divided into high and low groups. There were 65 cases with a high index, and 35 with a low. Of 29 children less than 16 years of age in the series, 23 (79%) fell into the high index group, and only 6 (21%) into the low. The marrow eosinophilia was not mirrored in the peripheral blood.

Of the 65 patients with a high index, 54 (83%) were cured (45% spontaneously). More significant prognostically, none of the splenectomized patients died. Of the 35 patients with a low index, 16 (46%) were cured (23% spontaneously) and 8 (44%) of the splenectomized patients died of their disease despite surgery. Nine per cent of the patients with a high index died and 43% of those with a low index.

In patients with a high index the return of platelets to normal levels usually occurred in from a few days to a few weeks. A relapse was never observed once spontaneous recovery had occurred. There was a uniformly beneficial effect from splenectomy and an insignificant operative mortality rate. Patients with a low index had a lower rate of spontaneous recovery, a high incidence of malignant purpura, a higher mortality rate, and frequent failure of splenectomy. 9 references. 1 figure. 6 tables.

Thrombocytopenia in Childhood. *Svend Heinild, Copenhagen, Denmark and Maj. Levander Lindgren, Stockholm, Sweden.* Acta pædiat., Suppl. 77: 149–53, 1949.

A followup study was made of all the cases of thrombocytopenic purpura registered during a 25-year period in pediatric clinics in Copenhagen, Gothenburg, Stockholm and Uppsala. The series comprised 113 patients, and is the largest one published so far. The interval of observation ranged from 1 to 28 years, average 9.4 years. Review of the data led to the following classification of the cases:

		Died
I. Infectious (or postinfectious) thrombocytopenia	63	13
II. Systemic diseases	9	7
III. Acute essential thrombocytopenia	28	1
IV. Chronic essential thrombocytopenia	13	0
Total	113	21

I. *Infectious (Postinfectious) Thrombocytopenia.* This group included all cases in which an infection could be demonstrated immediately before the first appearance of symptoms (as a rule, 1 to 2 weeks earlier). The ages at first appearance as related to outcome were as follows:

Age at onset (yrs.)	0–2	3–5	6–8	9–11	12–14
No. of cases	20	23	7	10	3
Deaths	10	1	1	1	0

In infancy, in which the infection often has the character of sepsis, the mortality is alarmingly high. The 50 living patients were clinically well when re-examined. Six had subnormal platelet counts (< 150,000) although with normal bleeding time.

II. *Systemic Diseases with Thrombocytopenia.* This group included only those cases in which the diagnosis of thrombocytopenic hemorrhagic purpura was actually recorded in the hospital records. The diagnoses of aplastic anemia, leukemia, megaloblastic anemia, malignant tumor, hepatosplenomegaly or splenomegaly were also made. The mortality rate was high.

III. and IV. *Essential Thrombocytopenia, Acute and Chronic.* This group included all cases in which no etiologic factor could be demonstrated. The clinical picture did not differ from that encountered in drug sensitivity or in the benign infectious type. The acute form of essential thrombocytopenia is cured definitively after 1 to 6 months. The chronic form has an intermittent course with symptom-free periods of up to 7 years. Recurrence often seems to appear in connection with an infection. The age distribution of the cases was as follows:

Age at onset (yrs.)	0–2	3–5	6–8	9–11	12–14
No. of cases	10	15	4	6	6
Deaths	1	0	0	0	0

At the followup examination every acute case had a normal platelet count, whereas some of the chronic cases showed subnormal values (< 150,000). In 2 the bleeding time was prolonged. All were free from symptoms.

Splenectomy. Splenectomy was performed in 12 cases, with the following results:

Diagnosis	No.	Recovery	Relapse	Died
Infectious thrombocytopenia	3	1	1	1
Essential acute thrombocytopenia	1	1	0	0
Essential chronic thrombocytopenia	4	3	1	0
Splenomegaly	2	1	1	0
Leukemia	2	0	0	2

The merit of splenectomy in the thrombocytopenic purpura of childhood is still controversial, except in those cases with rapidly progressive hemorrhagic phenomena in which death or severe catastrophe seems imminent. One cannot evaluate the circumstances leading to death from

the published figures in the above series of patients; it is possible that some of these deaths would not have occurred had splenectomy been done. Nevertheless, the results here reported indicate that the long-range prognosis for recovery, probably complete but certainly asymptomatic, is good without splenectomy in both the postinfectious and the idiopathic varieties of thrombocytopenia in childhood, provided the initial attack is survived. The statistics bring out also that the majority of childhood cases are in children under 5 years of age, that acute cases are more common than chronic ones, and that deaths when they occur are almost always in infants and children less than 3 years of age.

The discussors of this paper at the time of its reading spoke of the frequency of purpuric attacks in the newborn period, and the fact that in most of these infants the symptoms have been not serious. In evaluating thrombocytopenia in the newborn one must remember that the platelet count is normally lower in the first few months of life than later on.—EDITOR.

Blood and Bone Marrow Studies in Renal Disease. *Irwin R. Callen and Louis R. Limarzi, University of Illinois College of Medicine, and the Research and Educational Hospitals, Chicago, Ill.* Am. J. Clin. Path. 20: 3–23, Jan. 1950.

The literature is first reviewed. An analysis is then given of the peripheral blood and bone marrow findings in 102 patients with nephritis or related diseases. Anemia was rarely present in these cases unless there was an associated elevation of the nitrogenous waste products in the blood. Anemia was not related to proteinuria, was not present in nephrosis without azotemia, and was not correlated with damaged kidney function per se.

The bone marrow was hypercellular in 80% of patients with renal disease and azotemia. Hypercellularity involved mainly the myeloid and megakaryocytic cells. Erythropoiesis was normal, as a rule. Quantitative and qualitative hypoplasia of the erythroid tissue was observed only when the nonprotein nitrogen level of the blood was over 150 mg. per 100 ml. Aplasia of the normoblastic tissue was never observed. A rather common observation was the presence of frequent numbers of eosinophils in various stages of maturation.

The discrepancy between the anemia in the peripheral blood and the apparent adequacy of the erythroid tissue in the bone marrow in many cases of chronic glomerulonephritis is difficult to explain. The mechanism regulating the delivery of cells to the blood stream appears to be at fault, rather than the maturation of the cells. Even when anemia was present there was no interference with leukopoiesis and megakaryopoiesis. A cellular bone marrow in chronic glomerulonephritis is not in itself evidence of hemopoietic activity nor does it have prognostic significance.

The bleeding state commonly observed in the terminal stage of chronic nephritis or uremia seems not to be due to injury of megakaryocytes in the bone marrow, since the platelets in the blood are adequate. The bleeding seems due to a combination of toxic capillary injury and prothrombin deficiency, both resulting from the toxic retention products associated with the abnormal renal condition. 83 references. 4 figures. 6 tables.

Infectious Lymphocytosis. *B. K. Lemon and D. H. Kaump, Provident Hospital, Detroit, Mich.* J. Pediat. 36: 61–68, Jan. 1950.

Infectious lymphocytosis is a benign process characterized principally by marked leukocytosis and absolute lymphocytosis. A study regarding this type of leukocyte response in children was initiated when a 29-month-old infant was hospitalized because of a fracture of the tibia. This child had an initial leukocyte count of 48,000 per cu. mm. with 88% lymphocytes. The leukocyte count rose to 70,400 per cu. mm. with 80% lymphocytes before regressing to normal. Because of these findings a survey was made of 49 children at the Sarah Fisher Home from which this child had come. All but 3 were in the 2-year and 3-year range. Seventeen were found with a total leukocyte count over 15,000 per cu. mm., with a differential of over 70% lymphocytes or eosinophils of over 3%. Nineteen had a total leukocyte count of less than 15,000 but with abnormal lymphocyte or eosinophil percentages. Thirteen had these hematologic values within normal range. No infectious mononucleosis or liver disease could be demonstrated.

Hematologically, the disease is characterized by an absolute leukocytosis and lymphocytosis of varying degrees which may persist for from 4 to 7 weeks in the acute and from 7 to 12 weeks in the subsiding and late phases. The lymphocyte morphology is not abnormal. Large mature cells tend to predominate in the phase of greatest leukocytosis and again in the phase immediately following recovery as indicated by a return to normal of the total leukocyte level. Eosinophilia is frequent in the recovery phase. The bone marrow contains a high percentage of lymphocytes of normal mature morphology, thus distinguishing infectious lymphocytosis from infectious mononucleosis and lymphatic leukemia.

The leukocyte count and careful morphologic study are the only dependable diagnostic criteria. The disease should be thought of when young children manifest a persistent rhinitis and low-grade fever and have a paucity of other signs or symptoms. Other clinical manifestations reported by others may include vomiting, diarrhea, irritability, abdominal signs and symptoms, central nervous system signs and symptoms, and transient dermatitis. The disease is benign and self-limited. Sulfadiazine and penicillin had no beneficial effect in these patients. 18 references. 1 table.

Eosinophilic Leukaemia and Familial Eosinophilia. Two Illustrative Cases. *J. D. Gray and Sydney Shaw, London, England.* Lancet. 6590: 1131–34, Dec. 17, 1949.

Of 23 published cases of eosinophilic leukemia, 18 occurred in males, with a tendency to an acute rather than a chronic course. In most cases the peripheral blood showed mature eosinophils. A boy aged 1 year and 4 months is described, who had rapid enlargement of the liver and lymph glands. The peripheral blood showed 20 to 50% eosinophils which were mature; the bone marrow showed an increase in eosinophils and eosinophil precursors with some atypical premyelocytes. These showed an indented pale staining nucleus with a few large coarse mixed eosinophil and basophil granules at the indentations. There was no evidence of eosinophilia or allergic symptoms in the family.

The literature on familial eosinophilia is briefly reviewed: Fourteen families have been hitherto reported, with transmission occurring through the mother in 11. At various times the liver, spleen and lymph glands enlarge temporarily in observed members of the families; the marrows show an eosinophilic preponderance with orderly progressive eosinophil maturation. All known causes of eosinophilia should be ruled out.

A boy aged 2 years and 4 months showed a peripheral blood eosinophilia of 59%. The marrow showed a considerable increase in eosinophils with absence of primitive leukocytes. There was eosinophilia in his mother and her sister. The patient had had attacks of bronchitis and wheezing, and his mother suffered from "catarrh." While under observation there was no progression of signs or symptoms.

Allergy is commonly noted in familial eosinophilia. This may be due to the eosinophilia and not that the eosinophilia results from the allergy. The infiltrative power of the eosinophil is stressed, as seen in cases of eosinophilic leukemia, and as has been noted in operations and biopsy specimens from patients with familial eosinophilia. 17 references. 5 figures.—*Author's abstract.*

Aminopterin in the Treatment of Acute Leukaemia. *J. V. Dacie, D. L. Mollin, E. Dresner and J. C. White, London, England.* Brit. M. J. 1: 1447–57, June 24, 1950.

The results of treatment in 13 cases of subacute and acute leukemia in children and adults are reported. Details of treatment in 8 cases are given in full. The results in the 13 cases were as follows:

Adults. Myeloblastic leukemia: 5 cases: No improvement in 4, temporary remission in one. Monocytic (Schilling type) leukemia: 1 case: temporary remission.

Children. Myeloblastic leukemia: 3 cases: temporary remissions in all. Lymphoblastic: 4 cases.

Remissions followed treatment in all of 7 children and in 2 of 6 adults. In children these took the form of almost complete clinical restoration to normality and extensive though not complete disappearance of leukemic cells from the bone marrow. Clinical relapse was preceded by the reappearance of blast cells in the bone marrow, and later in the blood. The duration of remissions varied from a few weeks to several months. In the adults the remissions were of 3 to 5 weeks duration only.

The dosage of aminopterin used was 0.5–1.0 mg. daily for children and 1–3 mg. daily for adults. Treatment was continued until there was a satisfactory hematologic response or toxicity compelled withdrawal of the drug. The intramuscular route was used in the earlier cases; later the drug was found to be just as active when given by mouth and in all further cases drug was given orally. After the initial course of aminopterin, further courses were given as required by occurrence of relapses. It was found necessary in most cases to administer the drug to the stage of toxicity to obtain an hematologic response. The chief toxic manifestations were ulcerative stomatitis, purpura, bleeding, diarrhea, leukopenia, marrow aplasia, vomiting and alopecia. Severe toxicity necessitates withdrawal of the drug and the administration of antibiotics and blood transfusions. In the present series, blood was transfused only when necessary to replace hemoglobin and red cells lost by hemorrhage, etc. and was unlikely, in the amounts given, to have had any effect in procuring the remissions obtained.

The effect of aminopterin on leukemic cells was studied by serial marrow biopsies. Primitive, undifferentiated cells appeared to be the most affected. Their disappearance from the blood and marrow was associated with a fall in the number of mature granulocytes, due to destruction of their precursors. Leukemic lymphoblasts were also affected. In patients who responded favorably, disappearance of primitive leukemic cells was associated with a spontaneous rise in red cells and platelets and a rise in the peripheral leukocyte count to normal levels, the cells being normal adult forms. At this stage, the bone marrow may appear normal, with active erythropoiesis and leukopoiesis but, in the authors' series, careful search revealed a slight excess of blast cells in all cases.

When treatment was given for the relapses which inevitably ensued, it was apparent that the leukemic tissue became more and more resistant to aminopterin, this resistance being restricted to the leukemic tissue and not a general cytological resistance, as toxic manifestations in the gut, skin, etc. followed as readily. The cause of this acquired resistance is unknown—whether persistence of originally resistant strains of leukemic cells, mutation, or altered metabolic requirements.

The possible modes of action of aminopterin and other folic acid antagonists in leukemias, and the indications for their uses, are discussed. 24 references. 13 figures.—*Author's abstract.*

Serum Iron and Iron-Binding Capacity of the Serum in Children with Severe Mediterranean (Cooley's) Anemia. *Carl H. Smith, Thomas R. C. Sisson, William H. Floyd, Jr. and Shirley Siegal, New York Hospital and Cornell University Medical College, New York, N. Y.* Pediatrics 5: 799–807, May 1950.

The serum iron and latent iron-binding capacity were determined in 15 children with severe Mediterranean anemia, the majority of whom were patients in an ambulatory transfusion clinic. For control purposes similar measurements were made of 11 normal children (aged 2 weeks to 10 years), and 10 adults (aged 22 to 67 years). Measurements were also taken of 10 adults with the trait of Mediterranean anemia (aged 27 to 35 years), of 8 children with the trait and mild type of Mediterranean anemia (aged 2½ to 12 years), and of 2 children with iron deficiency anemia (ages 15 and 19 months). The mean values and standard deviations, or ranges of spread, are summarized in the accompanying table.

In children with severe Mediterranean anemia the iron-binding component of the serum is fully saturated with iron instead of being approximately one-third saturated, as occurs normally. This is less consistent in the asymptomatic and mild forms of the disease. It is suggested that the continuous destruction of erythrocytes, perhaps from birth, leads to excessive liberation of iron, to its deposition in various organs, and to full saturation of the iron-binding protein. The occurrence of similar findings in some persons with the mild or trait form of the disease implies that prolonged and insidious hemolysis may occur in the asymptomatic individual but that compensatory hematopoiesis prevents the development of anemia. The marked reduction in total iron-binding capacity suggests that a congenital and possibly transmissible deficiency of the metal-binding Fraction IV-7 of plasma occurs in Mediterranean anemia. This possibility is now being explored.

The two children with iron deficiency anemia had a reduction in percentage saturation of iron-binding protein (16.9 and 12.8%), an increased level of unbound latent iron-binding capacity (480 and 490 γ), and a markedly elevated total iron-binding capacity (590 and 581 γ).

The high serum iron and absence of latent iron-binding capacity in the children with severe Mediterranean anemia receiving multiple transfusions correspond to the changes regarded as diagnostic of transfusion hemosiderosis and hemochromatosis. While these changes remain unaltered in Mediterranean anemia, their reversibility has been demonstrated in 2 children with hemophilia who received weekly transfusions of small amounts of blood. After cessation of this therapy there occurred a decrease in serum iron and a gradual restoration of iron-binding capacity. 15 references. 6 tables. 1 chart.

	Serum iron content	Latent iron-binding capacity (gamma/100 ml.)	Total iron-binding capacity (gamma/100 ml.)	Degree of saturation (%)
Normal Adults and Children	155.7 ± 31.2	255.86 ± 54.7	411.56 ± 65.4	38.0 ± 7.2
Normal Adults	162.88 ± 15.7	201.27 ± 44.9	364.15 ± 51	45.3 ± 5.9
Children with Severe Mediterrenn Anemia	269.6 ± 62.9	0	269.6 ± 62.9	100%
Adults with Trait (range)	126 – 404.5	0 – 337.5	126 – 521	34 – 100%
Children with Trait	151.5 – 384.9	0 – 425	151.5 – 614.4	30.8 – 100%

Essential Lymphocytophthisis. A New Disease Picture in Infant Pathology (*Essentielle Lymphozytophthise. Einneues Krankheitsbild aus der Säuglingspathologie. Vorlaufige Mitteilung*). *Eduard Glanzmann and Paul Riniker, Bern, Switzerland.* Wien med. Wschr. 100: 35–36, Jan. 1950.

This hitherto undescribed disease was observed in two infants, a girl of 4 months and a boy of 5 months. The infants were both dystrophic and had attacks of fever with short remissions, besides an extensive oidomycosis. The lips, tongue, buccal mucosa, soft palate and uvula were all heavily coated with a whitish exudate which also extended over the pharynx and deep into the larynx. The infants were hoarse, and suffered from occasional vomiting and dyspeptic stools. Besides the mycotic syndrome, there was also a sort of seborrheic dermatitis with impetiginous efflorescences, and during the later stages patches of measles-like exanthema, occasionally fading only to recur. Gradually the lungs became involved in a progressively extending bronchopneumonia that resisted all treatment. The cervical lymph glands were swollen but there was no splenomegaly. Blood examination revealed a lymphocytophthisis of the peripheral blood which was almost total. One infant died at the age of 5 months and the other at the age of 7 months, with signs of heart failure, pulmonary emphysema and edema, stupor and general convulsions.

Autopsy revealed similar changes in the two infants, namely, an oozing erythema, anemia, and mycosis involving not only the regions mentioned, but also the stomach and small and large intestines. There were several small sharply circumscribed ulcers in the ileum. Effusions were found in the abdominal, pleural and pericardial cavities with advanced lobular pneumonia, in one case with abscess formation and fibrinous pleurisy. The liver and kidneys were slightly swollen, the spleen was small, and the adrenal glands were normal. The marrow of the vertebrae and ribs was bright red. Histologic examination revealed an almost total absence of lymphocytes in the lymph glands,

Peyer's patches and spleen, leaving only a heavy mesh of reticulum cells. The death of a sister of one of the patients from somewhat similar symptoms suggests a familial incidence of the condition which is probably due to a constitutional weakness of the blood-forming organs. 1 figure.

Analysis of Fifty Cases of Erythroblastosis Fetalis. *Victor Ginsberg and Felix Feldman, Kings County Hospital, Unity Hospital, and Brooklyn Women's Hospital, Brooklyn, N. Y.* Am. J. Obst. & Gynec. 59: 618–26, March 1950.

This is an analysis of 50 patients with erythroblastosis fetalis treated in the past few years. Of these, 45 seemed due to Rh incompatibility, and 5 to ABO incompatibility. There were 26 boys and 24 girls. Deaths were evenly divided, 7 boys and 7 girls. These figures are at variance with statements that morbidity and mortality are more common among male infants. Ten were Negro infants, the number being roughly proportional to the percentage of Rh-negative individuals in that population.

There were 8 stillborns. Most of these mothers had previously had other severely jaundiced or stillborn infants. The maternal antibody titers ranged from 1:32 to 1:512. Of 23 infants treated with multiple transfusions, 6 died. Of 11 treated with exchange transfusion, none died. Of apparently milder cases given no therapy, 1 died. The hospital stay of the infants treated without exchange transfusions varied from 7 to 32 days.

Of 11 patients given exchange transfusions, the first 3 were treated via the saphenous vein and radial artery. These required long periods of hospitalization chiefly because secondary infections developed. The remaining transfusions were given via a plastic catheter inserted in the umbilical vein. The infants usually had a smooth recovery. Maternal antibody titers in this group ranged from 1:16 to 1:512. Blood counts, with three exceptions, remained within normal limits. Jaundice disappeared rapidly. The average hospital stay of the 8 babies transfused via the umbilical vein was only 8.7 days. Four of these mothers had had previous stillborns, but their infants, treated with exchange transfusion, survived.

It was concluded that decision as to mode of therapy cannot usually be made until after birth of the infant. Then, all the laboratory and clinical evidence must be evaluated. Exchange transfusion at present is the most effective way to treat severe erythroblastosis fetalis. The method of choice is by plastic catheter via the umbilical cord. 25 references. 5 tables.

The Treatment of Erythroblastosis Fetalis by Transfusion with Sedimented Red Cells. *Samuel Pennell, Maimonides Hospital, Brooklyn, N. Y.* Blood 5: 107–22, Feb. 1950.

The author has studied the results of the treatment of 28 infants

with erythroblastosis fetalis by transfusion of compatible sedimented red cells from bank blood. Twenty of these cases were attributed to Rh incompatibility, and 8 to ABO incompatibility. The indication for transfusion was taken as a hemoglobin level of 15 Gm. or less per 100 ml. of blood in any erythroblastotic infant within 36 hours after birth. Transfusion of sedimented red cells in 50 to 60 cc. amounts seemed adequate to raise the hemoglobin values with a minimum of load on the infant's circulation. Three of these patients died.

This method is recommended as superior to exchange transfusion because of its greater ease and simplicity, the ready availability of bank blood, and the minimal amount of anticoagulant contained in it. The amount of adult plasma carried along with the red cells is deemed too small to cause activation of any agglutinins which might be present in the infant. 24 references. 5 tables. 1 figure.

The results here reported are being quoted as showing that exchange transfusions with all their complexity are no longer needed in erythroblastosis fetalis, so long as blood bank erythrocytes are available. Survey of the data given in the tables accompanying the text of this report makes it clear, however, that well over 50% of the patients cited had erythroblastosis in mild or moderate form, while only a minority were severely affected. Since the benefits of exchange transfusion lie in the realm of the severe and critical cases, it does not seem reasonable to make comparisons between the two methods of treatment on the basis of less than a dozen severe cases treated with sedimented red cells, of whom three died!

Erythroblastosis fetalis is frequently treated with intravenous infusions of sedimented erythrocytes. These are given either mixed with a little donor plasma or after washing with saline, with the same precautions for compatibility as with whole blood transfusions. It is pointed out that the anemia can be corrected by a lesser volume of replacement fluid; the blood volume is not raised as much, thereby avoiding circulatory failure. It is also claimed that red cell infusions contain a minimum of anticoagulant substances and agglutin-accelerating and related sensitizing factors which may react harmfully with the already sensitized cells of the infant. At the present writing, however, these advantages are viewed as not fully substantiated by empirical observations. It has been argued, in rebuttal, that the plasma contained in whole blood transfusions is a corrective against shock symptoms, and is helpful in raising the lowered blood protein level.—EDITOR.

4. Cardiovascular System

Cardiac Anomalies in Mongolism. *Philip Rainsford Evans, London, England.* Brit. Heart J. 12:258–62, July 1950.

Congenital heart disease was found in nearly half of a series of mongoloid imbeciles dying in the first five years of life. In the records of 63 autopsies, 28 examples were found.

Ventricular and atrial septal defects each accounted for one-third of the individual lesions, and patent ductus arteriosus for one-sixth. Pulmonary stenosis and persistent atrioventricularis communis each occurred in 4 patients.

The Following Table Shows the Individual Anomalies Occurring 2 or More Times in the Hearts of 28 patients with Mongolism, or 4 or more Times in 50 Patients with Congenital Heart Disease Who Were Not Mongoloid

Lesion	Mongoloid		Not mongoloid	
	No.	Percentage	No.	Percentage
Auricular septum, all defects	17	60	18	36
Auricular septum, ostium primum or secundum, without persistent atrioventricularis communis	12	43	15	30
Persistent atrioventricularis communis	4	14	0	0
Ventricular septum, all defects	17	60	26	52
Ventricular septum, absent or nearly so	1	4	5	10
Ventricular septum, membranous defect without persistent atrioventricularis communis	12	43	21	42
Patent ductus arteriosus	8	29	20	40
Truncus arteriosus	0	0	4	8
Pulmonary stenosis	4	14	3	6
Coaretation of aorta	0	0	4	8
Transposition of aorta and pulmonary artery	0	0	7	14

Compared with other children with congenital heart disease dying in the same hospital in the same period, transposition of the great vessels, coaretation, and truncus arteriosus seemed to be unassociated with mongolism. Atrial septal defects and pulmonary stenosis were more common in the mongoloid patients than in the other patients. Speaking generally, the cardiac anomalies in mongolism appeared to be due predominantly to defects occurring in the earlier stages of cardiac development. 14 references. 5 tables.—*Author's abstract.*

5. Chemotherapy, Drugs, Poisons, Physical Agents

Clinical Observations on a New Sulfonamide (Gantrisin®). *John A. Bigler and Orville Thomas, Children's Memorial Hospital, Chicago, Ill.* Am. J. Dis. Child. 79: 785–90, May 1950.

Gantrisin®, 3-4, dimethyl-5-sulfanilamido-isoxazole, formerly known as NU-445, is a new sulfonamide which has a high degree of solubility and a low degree of toxicity. Because of its solubility there is no crystallization in the urine except in much higher doses than would be used therapeutically.

Dosages used in this study were 1 to 1½ Gr. per pound of body weight, divided into four or six daily doses. Blood levels varied from 3 mg. to 15.7 mg. %. The average blood levels varied from 6.4 to 10.7 mg. %. There were no toxic reactions or instances of leukopenia in the group. Crystalluria or microscopic hematuria did not occur.

One hundred twenty-one children with the usual infections of childhood, such as lobar pneumonia, bronchopneumonia, bronchitis, otitis

media, tonsillitis, adenitis and urinary tract infections were treated with this drug. Seven did not respond to treatment. The results were good in all remaining cases except in seven others which included 3 of atypical pneumonia, 1 of nonhemolytic streptococcal pyuria, 1 of rheumatic fever, 1 of infectious mononucleosis and 1 of roseola infantum. In all of the other cases there was a drop in temperature within 12 to 36 hours with marked clinical improvement.

The use of this drug compared favorably in infections of children with that of other commonly used sulfonamides. It has the added advantage of low toxicity, increased solubility and less crystalluria and hematuria. 7 references. 1 table—*Author's abstract.*

Sulfonamide Dosage in Early Infancy. *Julius B. Richmond, Harvey Kravitz and William E. Segar, Chicago, Ill.* J. Pediat. 36: 539–49, May 1950.

Blood sulfonamide levels appear to be maintained at a higher level for a longer period in infants ranging in age from the newborn to 2 months than blood levels obtained under comparable conditions in older infants. In general the results with subcutaneous administration are more favorable and more readily reproducible than those obtained with higher doses of orally administered drugs. This difference is presumably due to fecal loss of the drug with oral administration.

The subcutaneous administration of 0.032 Gm. per pound of body weight of a 5% mixture of equal parts of sodium sulfadiazine and sodium sulfamerazine in distilled water once every 24 hours maintained adequate therapeutic blood levels in infants under 2 months of age. The elevation of the dosage to 0.048 Gm. per pound of body weight similarly administered effected higher blood levels in a similar age group. In addition, the infants over 2 months of age maintained therapeutic levels for 24 hours on this increased dosage in many instances.

The oral administration of sulfadiazine as an initial dose of 0.13 Gm. per pound of body weight followed by 0.1 Gm. per pound every 24 hours maintained therapeutic blood levels in most instances in infants under 2 months of age. Slightly lower dosage was less effective. Because of the relatively larger dosage and greater variability of results, this route of administration is not suggested as preferable to subcutaneous administration in lower dosage as indicated above.

The better maintenance of blood levels following single doses of sulfonamides in young infants as observed in this study is attributed largely to the immaturity of renal function during this age period. 21 references. 4 figures. 1 table.—*Author's abstract.*

Death Following Intoxication by Sulfonamides. (*Dood tengevolge van intoxicatie door sulfonamiden*). *The Sie Kian, Utrecht, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 190–95, June 1949.

A 3-year-old boy who had previously received treatment for a derma-

tosis with small quantities of sulfonamides four times in four months, swallowed eleven tablets of a sulfonamide. Within four hours he became cyanotic, comatose and catatonic and developed a morbilliform eruption. Alkalinization treatment was given and seemed successful until the sixth day. Then fever, hemorrhage from the nose and the skin and edema began. Death soon followed. The blood before death showed leukopenia and eosinophilia (23%). The bone marrow had many megakaryocytes. Autopsy revealed hemorrhages into all organs, interstitial nephritis, and interstitial hepatitis. 12 references. 2 figures.—*H. Bartels.*

Clinical Observations on the Use of Terramycin Hydrochloride. *Ernest Q. King, Charles N. Lewis, Henry Welch, Eugene A. Clark, Jr., John B. Johnson, John B. Lyons, Roland B. Scott and Paul B. Cornely, Washington, D. C.* J.A.M.A. 143: 1–4, May 6, 1950.

Terramycin hydrochloride was administered orally to 30 patients with various diseases including pneumonia, urinary tract infections, whooping cough, bacteremia due to *Salmonella*, and lung abscess. The dosages were: in patients 14 years old or older without urinary infections, 750 mg. every six hours; with urinary infections, 500 mg. every six hours; in children 9 years old or younger, 500 mg. every four hours. The drug was found in the blood and urine within one hour and for five hours after administration. Side reactions were few and mild, except in one patient who had severe gastrointestinal distress with diarrhea and the drug was discontinued. One patient with severe pulmonary infection accompanied with pulmonary edema and avitaminosis died. In the remaining 29 patients, results were excellent in 9, good in 15, fair in 4 and poor in 1 only. The findings indicate that terramycin is a useful addition to the antibiotic drugs. 4 references. 1 table.

Paramethadione.—Paradione (Abbott) $C_7H_{11}NO_3$. Report of Council on Pharmacy and Chemistry. J. A. M. A. 143: 970, July 15, 1950.

The actions of paramethadione are similar to those of trimethadione, but the drug may be quantitatively less active. Paramethadione is indicated in the treatment of petit mal epilepsy, and other conditions for which trimethadione is used. Paramethadione is effective in a significant number of patients not benefited by trimethadione. The reverse is also true.

The side reactions resulting from paramethadione therapy are those produced by trimethadione, except that the incidence of photophobia and rash is diminished. The most serious side effect, as with trimethadione, is severe leukopenia, which occurs occasionally. White blood cell counts should be made bi-weekly during the first two months of therapy and at monthly intervals thereafter.

The dosage for adults is 0.9 Gm. daily, initiated in divided doses. Thereafter, the dose should be increased or decreased to provide the smallest dose which will just control the symptoms. For infants, the initial daily dose should be 0.3 Gm.; for children 2 to 6 years of age, 0.6 Gm. in divided doses.

Paradione is marketed in 0.3 Gm. capsules, and in 50 cc. dropper bottles holding a 65% alcoholic solution containing 0.3 Gm. of paramethadione in each cc.

Pulmonary Symptoms After Drinking Kerosene (*Longafwijkingen na het drinken van petroleum*). *J. J. Soer, Dordrecht, The Netherlands.* Maandschr. v. kindergeneesk. 17: 118–21, 1949.

After drinking kerosene 3 of 4 children showed pulmonic symptoms. A few rhonchi were heard which disappeared in a few days. Roentgenograms showed infiltration in the lower lobes around both hila. In 1 patient these features were already visible three hours after drinking the kerosene. The roentgenologic evidences disappeared in two to five weeks. 5 references. 2 figures.—*J. I. de Bruijne.*

Ingestion by Children of Various Substances, Including Poisons. *A. L. King, Oakland, Calif.* Permanente Found. M. Bull. 8: 20–28, Jan. 1950.

A listing is made of the substances ingested by children seen in the Permanente Hospital Emergency Room, Oakland, California, from 1945 to 1949.

There was a total of 196 cases of children ingesting poisonous substances. Ant poison containing arsenic was ingested by 37 children. Aspirin accounted for 17 of the ingestions. Other common articles ingested were barbiturates and thyroid tablets, each 6 children; lye, carbon tetrachloride, kerosene, matches and crayons, each ingested by 5 children.

There were 33 children admitted to the hospital because of ingesting poisons. One died of aspirin poisoning and manifested hyperpnea, acidosis and oliguria. The other child who died had swallowed lye, and, although early dilatation of the esophagus was carried out, esophageal strictures, necrotizing esophagitis, esophagoperitoneal fistula and peritonitis developed in about a month.

Three case reports of poisoning due to less well known poisons are included: Tung nuts (the oil from which is used in making varnish) caused an acute, severe gastroenteritis. Ingestion of privine nose drops (0.1%) led to acute, severe hypertension, 210 mm. systolic and 160 mm. diastolic, and stupor in a 19-months-old white boy. Magnesium sulfate intramuscularly seemed to be effective in reducing the blood pressure. Naphthalene (moth balls) ingestion was followed by severe hemo-

globinuria, anemia and gastroenteritis with tarry stools, and the patient was cured by multiple transfusions. Of 93 non-poisonous foreign bodies ingested, 30% were coins and passed through the gastrointestinal tract without difficulty unless held up in the esophagus.

Probably the most useful bit of knowledge about a poisoned child is to know, preferably in chemical terms, specifically what has been swallowed. Lack of this knowledge sometimes leads to giving dangerous antidotes to children who have swallowed innocuous substances. 7 tables.—*Author's abstract.*

Boric Acid as a Poison. Report of Six Accidental Deaths in Infants. *E. Gordon Young, R. P. Smith, and O. C. MacIntosh, Halifax, N. S.* Canad. M. A. J. 61: 447–50, Nov. 1949.

The deaths of 6 babies, 6 to 11 days of age, are reported as the result of one feeding of milk which had been diluted with a solution of boric acid (2.5%) in error for sterile water. The deaths occurred in from nineteen hours to five and one-half days.

The clinical diagnosis of death was intoxication with respiratory failure. Three autopsies were done and the pathologic diagnosis was gastroenterocolitis, hepatitis, nephrosis, and cerebral edema and congestion, of toxic origin. In 1 patient, the longest to live, was bronchopneumonia with nephrosis. All showed an exfoliative dermatitis. The chemical examination established the presence of boric acid in the fluid used as a diluent of the milk, in the stomach, and, by quantitative analysis, in brain and liver. A concentration of 0.049% (total 61 mg.) was found in the liver and of 0.025% (total 123 mg.) in the brain of 1 child dying forty-five hours after the suspected feeding. By calculation, the amount of boric acid ingested must have been less than 3 Gm.

Associated symptoms which developed progressively in this group of infants were: (1) vomiting and diarrhea, increasing in intensity after the first day; (2) excoriations of the skin on the second and subsequent days; (3) convulsive movements, especially twitchings of the facial muscles and of the extremities; (4) cyanosis and collapse as the terminal picture. 7 references. 1 table.—*Author's abstract.*

6. Clinical Pathology

Blood Sugar Values in Premature Infants. *Mildred A. Norval, Mayo Clinic, Rochester, Minn.* J. Pediat. 36: 177–84, Feb. 1950.

The postabsorptive or fasting levels of blood glucose were measured in 33 normal premature infants during their first two weeks of life. In addition, 9 other premature infants were studied who died before the third day of life. Four of these had extremely low levels, but it is not known whether these values were primary or secondary to the infants' condition. A modified Somogyi micromethod was used for the evaluations. The technical error of the determination by the method used,

measured as the standard deviation, was 4.2 mg. per 100 ml. (7.0% of the mean). The mean blood glucose value for the entire group was 60.0 ± 0.56 mg. per 100 ml.; the range was from 15 to 117 mg. per 100 ml.; the variability, measured as standard deviation, 15.3 mg. per 100 ml. (25.5% of the mean).

Weight, body temperature, and feeding schedule of the infant did not influence the level conspicuously. No clinical signs of hypoglycemia were observed in the cases with low values, as judged by the intradaily standard deviation. There was no clear evidence that the value for the blood sugar becomes progressively stabilized during prematurity.

The mean value for blood glucose in these premature infants during the first week of life was lower by 4.7 ± 0.95 mg. per 100 ml. than the corresponding value previously obtained for fullterm infants at birth. During the first day of life serial blood sugar determinations showed rapid decline in the first four hours, and then slower decline to a minimum in 8 to 12 hours after birth. A slight rise usually occurred in the next 12 to 16 hours, even when the infants were not fed. The blood glucose rose progressively in the days following birth, leveling off at about the end of the first week of life. 21 references. 4 figures. 2 tables.

Staub Effect in Children. Studies of the Blood Sugar Regulation by Means of Double and Triple Glucose Tolerance Tests. *Olli Somersalo, The University of Helsinki and The Children's Castle, Helsinki, and the Kronprinsessan Lovisa's Children's Hospital, Stockolm.* Acta pædiat. Suppl. 78, 1950.

The so-called Staub effect refers to the observation that if two or more test doses of glucose are given to a fasting subject, the second and later doses induce a lower rise in the blood sugar level than did the first dose. The hyperglycemia which follows the first dose is believed to either (1) increase the insulin production, producing a surplus of it in the blood for the next doses, or (2) activate the insulin, which therefore exercises a stronger effect during the second dose, or (3) decrease the pituitary secretion with a resulting decrease in inhibition of insulin which therefore has a stronger effect during the second dose.

The author investigated the Staub effect in the pediatric age period. Twenty-seven intravenous double and triple glucose tolerance tests were performed on children 1 to 12 years old. The intervals between the injections varied from 40 to 200 minutes and the doses from 0.25 to 2 Gm. per Kg. of body weight. The curves were compared by using the criterion of length, i.e., the time return to 100 mg. per 100 ml. using the Hagedorn-Jensen micromethod on capillary blood. No Staub effect was ever noted if the second tolerance test was performed after the first curve returned to normal. In those experiments where the interval between injections exceeded 60 minutes, a slightly negative Staub effect was as a rule obtained—the second curve descended more slowly than the first.

Eight similar experiments performed on adults showed the Staub effect as reported in the literature, with the exception of one case, in which the first curve fell more rapidly than normal. Experiments in which the intervals between injections were long revealed a negative Staub effect, both in adults and children. In 22 double oral tolerance tests in children the Staub effect appeared as a rule, but there were 5 inverted reactions. This reversal was obtained particularly in those tests where the first peroral curve was unusually low.

The peroral double tolerance test was found to give an unreliable picture of the blood sugar regulation, since the tolerance tests are influenced by several variables, such as the evacuation rate of the stomach and the rate of absorption. If an intravenous dose is administered instead of a second peroral dose, and this intravenous curve is compared to a similar earlier one, a more exact record of the change in the blood sugar regulation induced by the peroral curve can be anticipated. A "standard" curve was obtained by administering an intravenous dose immediately before the peroral one. Thirty such intravenous-peroral-intravenous tests were performed on children aged 1 to 13 years. The second intravenous tolerance curve was as a rule shorter than the first, particularly when the peroral dose produced a very slight elevation of the blood sugar.

These observations showed that the intravenous glucose tolerance test does not accelerate the blood sugar regulation in children. An oral dose brought about this effect, although it produced a much slighter elevation of the blood sugar or none at all. It was concluded that the mechanism of the Staub effect is influenced by some other factor besides those mentioned above.

This factor presumably exercises its effect during the absorption of glucose or during its later passage through the portal circulation. It could be an increase in the minute volume of the liver, or else some blood sugar reducing substance elaborated in the intestine during the process of absorption. 147 references. 49 figures. 4 extended tables.

Urinary Corticoid Values of Children as Determined Chemically. *N. B. King and H. L. Mason, Mayo Foundation, Rochester, Minn.* J. Clin. Endocrinol. 10: 479–91, May 1950.

Corticoids were measured as formaldehydrogenic substances on periodic oxidation according to the method of Corcoran and Page, in children of various ages. A group of 95 children who were healthy, ranging in age from 2 days to 15 years and consisting of 32 boys and 63 girls was studied. The results are briefly summarized as follows:

Age	Corticoids (mg./24 hrs.) mean values
newborn	0.18
1–4 yrs.	0.23–0.24
5–10 yrs.	0.22–0.36
10–15 yrs.	0.29–0.45

There was a statistical increase of 0.01 mg. per year but overlapping was marked. After 10 years of age, the values approached adult figures. The differences between male and female were not statistically significant. These figures were somewhat lower than those obtained by phosphomolybdic reduction. Daily variations for any one individual were as great as ± 30% of the mean for that individual.

A second group of 14 hospitalized children was investigated. Two undergoing orthopedic surgery demonstrated rises in corticoids up to 2.3 times the highest normal for their ages, and returned to normal between the third and fifth postoperative days. A less marked rise was observed following two abdominal operations. Rises were also noted with fever (therapeutic in some) and infection. 7 figures. 12 references. 2 tables.—*A. M. Bongiovanni.*

It is difficult to decide on a method for "corticoid" measurements, since no one is particularly specific. Perhaps the method of Heard, Sobel and Venning (J. Biol. Chem. 165: 699, 1946) is less cumbersome and more reproducible.—EDITOR.

7. Endocrine System

A Study of the Content of Active Principle in the Human Thymus. (*Essais d'évaluation de la teneur du thymus humain en principe actif*). *J. Comsa, Paris, France.* Arch. franc. pediat. 7: 51–7, 1950.

Extirpation of the thymus in guinea pigs leads to arrest of growth, increased susceptibility to infection, debility, and death within 30 to 60 days. This is relieved by thymus extracts. The per cent of thymocytes in the cortex of the thymus among the other cells parallels closely the concentration of an active principle according to a method of assay used by the author. The thymocytes are fewer in infants dying of cachexia and infection than from sudden accidental causes. 10 references. 2 tables.—*A. M. Bongiovanni.*

See next abstract—EDITOR.

Investigations with Thymus Hormone Therapy in Athrepsia of Infancy (*Recherches sur l'opothérapie thymique dans l'athrepsie du nourrisson*). *J. Comsa, Paris, France.* Arch. franc. pediat. 7: 131–9, 1950.

The weight of the thymus in relation to total body weight averaged 2.5% in a group of 14 normal infants. In athreptic infants this was much less (about 1.0% or lower). The athreptic infant is compared to the guinea pig without a thymus in which growth is arrested and death finally ensues. Sixteen patients with athrepsia were treated with a thymus extract from calves prepared by Bezssonoff. Of these, 8 showed a sudden increase in weight and general improvement; 3 infants recovering from severe infections showed weight gain and improve-

ment with the extract; 3 debilitated newborn infants exhibited increased digestive tolerance and growth. Other thymus extracts were not as successful. 6 references. 2 tables.—*A. M. Bongiovanni.*

These ideas regarding thymus function are interesting and in need of confirmation or further evaluation.—EDITOR.

A Case of Ovarian Agenesis (*Een gebal ban ovariële agenesis*). *S. van Creveld and O. M. de Vaal, Amsterdam, The Netherlands.* Nederl. tijdschr. v. Geneesk. 93: 3311–19, Sept. 24, 1949.

The diagnosis of ovarian agenesis was made in a 14-year-old girl with a height of 132 cm. and a weight of 35 Kg. In addition to dwarfing she showed a webbed neck, lack of breast development, absence of axillary and pubic hair, and kyphoscoliosis. Except for osteoporosis and kyphoscoliosis, the bony development was normal. The urine contained an increased amount of gonadotropic hormone and a low content of 17-ketosteriods.

By periodic treatment with stilbestrol it proved possible to produce withdrawal bleeding regularly; the breasts developed slightly. No hair growth followed; this might be due to concomitant hypoplasia of the adrenal cortex. Since this adrenal-cortical insufficiency might be compensated by androgenic substances, the patient received 3 times a week, with a rest every fourth week, injections of 20 mg. testosterone propionate and 5 mg. estradiol benzoate suspended in 1 ml. of cod liver oil. Following a few months of this treatment, secondary hair growth appeared and the breasts developed. Withdrawal bleeding occurred regularly. The statural growth proceeded slowly and the general condition improved. 15 references. 3 illustrations. 1 graph.—*M. G. Stronk.*

Pubertas Praecox in a Girl of Rather More than Two Years and a Half. (*Pubertas praecox bij een meisje van ruim 2½ jaar*). *B. van Aken, Utrecht, The Netherlands.* Nederl. tijdschr. v. Geneesk. 93: 1325–29, April 23, 1949.

The diagnosis of precocious puberty was made in a 2½-year-old girl who had been menstruating for a few months. The breasts and labia were enlarged and there was pubic hair. Height, weight and ossification of the wrist corresponded with those at 4½ to 6 years. Differential diagnosis included: granulosa cell tumor, tumor of the ovary, primary chorionepithelioma, tumor of the adrenals, cerebral disorder, tumor of the pineal gland or hypothalamus, encephalitis, hydrocephalus and finally the constitutional type without any demonstrable tumor or endocrine disturbance. In this last form, the excretion of gonadotropin does not exceed that of puberty; the 17-ketosteriod excretion may be slightly increased. By elimination the diagnosis of constitutional precocity was made. The child was one of a binovular twin, the boy being

normal. In a paternal aunt development of the breasts had begun at 6 years of age. 10 references. 1 figure.—*Ph. Arons.*

The Adrenogenital Syndrome (*Het adrenogenitale syndroom*). *S. J. de Jong, Rotterdam, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 155–62, May, 1949.

Vomiting and dehydration in a 14-day-old boy were diagnosed as due to hypertrophy of the androgenous zone of the adrenal cortex. The condition improved with administration of NaCl and desoxycorticosterone. The patient is doing well at the age of 2 years but needs DOCA regularly. Slight evidences of precocious puberty are present.

A second patient, a pseudo-hermaphrodite, was hospitalized at the age of 16 days because of vomiting and dehydration. This infant also improved rapidly during administration of DOCA.

The diagnosis of hyperplasia of the androgenous zone of the adrenals with impairment of the other parts of the cortex was made in both patients by the finding of a low sodium chloride content of the blood and a high potassium level. The urine had an increased excretion of 17-ketosteroids, especially of those components derived from the adrenal cortex. 12 references. 1 figure. 3 graphs.—*M. G. Stronk.*

8. Eye, Ear, Nose and Throat

Recurring and Corneoscleral Varicella. *G. M. H. Veeneklaas, Utrecht, The Netherlands.* Ophthalmologica 119: 96–98, Feb. 1950.

In a family with 3 children, typical varicella developed in the youngest child when 7 months of age at a time when cases of the disease were occurring in the neighborhood. Neither of the other 2 children developed varicella at this time. About two months later, this child showed a recurrence of equally typical varicella after contact with a known case of the disease, and shortly after being vaccinated against smallpox. At about the same time in the oldest child, 5 years of age, who had been in contact with the same case, varicella developed, with typical symptoms except that a typical vesicle developed on the limbus corneae of the right eye, with a vascular band extending toward the medial corner of the eye. This vesicle flattened out into a small dark spot; the vascular band persisted for a longer time, and three years later a remnant of it still remained in the form of a very small, slightly raised stripe. Varicella developed in the other child in the family (2 and a half years old) about ten days after the oldest child; the disease ran a typical course except that a vesicle also developed on the limbus corneae of the right eye. As the occurrence of vesicles on the limbus corneae is unusual in varicella, its occurrence in 2 children of the same family suggests a hereditary disposition. 1 figure.

9. Gastrointestinal System

Secretin Tests with Bilumen Gastroduodenal Drainage in Infants and Children. *Gordon E. Gibbs, University of California Medical School, San Francisco, Calif.* Pediatrics 5: 941–46, June 1950.

A double lumen gastroduodenal tube was utilized by the author in studying the response of the pancreatic juice to the injection of secretin. When the tip of this tube is advanced into the duodenum, one channel drains that area, whereas the other has side openings for removal of the stomach fluid. The advantage gained is minimal dilution of the duodenal specimen by gastric fluids. Determination of the volume and composition of the pancreatic juice becomes more accurate. When the output of pancreatic fluid is scanty, as in many cases of pancreatic disease, the acidifying action of contaminating gastric juice may obscure the neutrality or alkalinity of the duodenal secretion and the weak enzymes which may be present, thereby making some pancreatic deficiencies seem complete.

Secretin (Wyeth) was injected intravenously, while this tube was in place, in 12 normal infants and children, 11 with cystic fibrosis of the pancreas, 2 with celiac disease and 1 each with lipoid pneumonia and Salmonella enteritis. The dosage given was one clinical unit per kilogram of body weight while the patient was sedated. The changes in content of trypsin and amylase and in *p*H and titrable alkalinity in the duodenal fluid were then measured.

In the normal subjects, whose ages ranged from 1 month to 11 years, the effect of secretin was to increase the average volume from 2.2 ml. to 10.3 ml./20 min. Without secretin, the *p*H averaged 7.4. After secretin the average was 8.4. The alkali titration increased correspondingly from 11 to 61 mEq. per liter. Trypsin concentration decreased from an average value of 79 to a value of 62, a 22% decrease. Omitting the normally low figures for the 6 infants, the amylase concentrations showed a 10% decrease. These decreases were more than offset by the increase in volume flow.

One significant feature of the use of secretin and the bilumen tube is the more uniform values in control patients. Narrowing of the normal range increases the usefulness of the test, since diagnosis of cystic fibrosis of the pancreas depends largely on the trypsin determination. Without secretin, the coefficient of variation of the trypsin concentration was 42%. With secretin it was 30%. Without secretin, the alkali titration was essentially negligible. With secretin an average value of 61 mEq./l. was obtained with a coefficient of variation of 26%.

In the 11 patients with cystic fibrosis of the pancreas, aged 2 months to 8 years, the volume of fluid obtained in 20 minutes before secretin averaged 2.3 ml. After secretin the volume was 1.9. This lack of increase after secretin is evidence that stimulation of bile flow or succus

entericus by the secretin or a concomitant gastrointestinal hormone does not confuse the tests done. The mean alkali titration went up from 4 to 7 mEq. per liter, essentially no change. The mean *p*H rose slightly from 7.1 to 7.6. No amylase or only faint traces were demonstrable. The trypsin concentrations and output per 20 minutes were almost negligible, before and after secretin. Two patients who had a trace of proteolytic enzyme in the preliminary period did not show it under the influence of secretin. Consideration of the volume, alkali, *p*H and amylase would tend to guard against the making of a false diagnosis of fibrocystic disease in the rare instances in which this disease is not present but where tryptic action is not obtained (due, possibly to lack of enterokinase).

In 2 children with celiac disease the volumes of juice without secretin injection were higher than in the normal controls, as has often been noted. The volumes rose after secretin, but less excessively. The 20-minute secretion rates were 6.5 and 13.0 ml. before secretion, 16.2 and 24.5 afterward. The trypsin figures were normal and responded normally. The amylase values were low, with no stimulation by secretin. The children with lipoid pneumonia and Salmonella enteritis gave essentially normal figures.

These investigations have thus demonstrated that bilumen gastroduodenal drainage is practical and useful in the small infant. This approach when combined with secretin seems to increase the reliability of duodenal drainage without adding significantly to the difficulties of performance. 20 references. 1 figure. 1 table.

The Frequency of Peptic Ulcers in Children (*Ulcussjukdomen hos barn särskilt med hänsyn till frekvensen*). *F. Karlström, Karlstad, Sweden.* Sven. läk. tidn. 46: 1218–23, 1949.

This paper deals with ulcers in children who have passed infancy and in whom the disease can be considered primary as in adults. Such ulcers are rare in childhood. A reported increase of the frequency during the last ten years is considered as due to improvement in diagnosis. Statistics from all hospitals in Sweden for the last five years revealed that 30 cases of peptic ulcer in children have been treated during that period. The age distribution was as follows:

Years:	3	6	8	9	10	11	12	13	14
No. of cases:	1	1	1	2	6	5	4	5	5

Total 30 (20 boys, 10 girls).

The symptoms of the disease in children do not differ from those common in adults. The results of conservative treatment are good. Hence surgery should not be used without strong indication of need. Probably many children are treated outside of hospitals, and many ulcers have perhaps escaped detection, so the disease may not be too

uncommon and ought to be kept in mind when digestive disturbances in children are being investigated. 22 references.—*G. Tunevall.*

Ulcerative Colitis in Children. *Rutger Lagercrantz, Stockholm, Sweden.* Acta pædiat., Supp. 75:89–151, 1949.

Following a survey of the literature the author presents clinical, prognostic and bacteriologic studies of 134 cases of ulcerative colitis in children. The following were the criteria for diagnosis: long period of illness (at least half a year and as a rule longer); purulent, bloody diarrhea; typical rectoscopic or x-ray findings; negative stool cultures. The disease seems to have become more common in children in Stockholm in recent years. The subjects comprise 78 boys and 56 girls. Most came from the upper social classes.

Psychic factors seem of importance in the development of the disease. A relatively large percentage had unhappy environments. Some relapses and deterioration in health seemed directly due to psychic causes. A few patients who were treated psychiatrically showed improvement. Further investigations of the psychosomatic aspects are needed. Most patients had normal delivery and birth weight and were breast fed. Prior to onset most had normal stools. A relatively large number apparently suffered from preceding infections of the upper respiratory tract. The majority became ill between the ages of 2 and 6. In 22 the onset occurred in conjunction with another disease, usually an infection. About 80% began with insidious symptoms starting with frequent loose evacuations. They were initially afebrile and became invalided gradually. In 14 (11%) the first symptom was bleeding on or after normal evacuation. In 11 (8%) the onset was acute, with a temperature of over 100° F. and severe diarrhea with blood and mucus. 69 references. 7 tables.

Acute Diarrheal Disorders of Newborn Infants: Differential Diagnosis. *Harold Abramson, New York, N. Y.* Am. J. Dis. Child. 36:698–711, April 1950.

This paper comprises a discussion of the acute diarrheal disorders of newborn infants as occurring in the nurseries of hospitals giving lying-in care. An attempt is made to differentiate the acute diarrheal disorders of newborn infants, and to present a working classification of these disorders, pending further studies of their causation. The cumulative literature was culled for such information as was pertinent to the problem. More especially, however, the differentiation was based on experience gained in the field in the investigation of outbreaks, and at the bedside. The differentiation is based mainly on clinical and epidemiologic grounds. Certain patterns of diarrheal disease are seen to emerge that warrant their separation as clinical entities from the general group of diarrheal disorders of the newborn.

The discussion is based on observations from 1934 to 1948, inclusive, of 321 outbreaks of acute diarrheal disorders that occurred in the nurseries of 91 hospitals giving lying-in care in New York City. Among the diarrheal disorders considered are: (1) epidemic diarrhea of the newborn; (2) epidemic nausea, vomiting and diarrhea of the newborn; (3) epidemic stomatitis and diarrhea of the newborn; (4) Salmonella infection of the newborn; (5) Shigella infection of the newborn, and (6) the unclassified diarrheas.

For the present, until simplified and appropriate laboratory procedures are devised, main reliance in diagnosis and differentiation must be placed on clinical and epidemologic features. 28 references. 2 tables. —*Author's abstract.*

A Study of Three Hundred Cases of Diarrhea in Infants and Children During the Summer and Fall of 1948. *Julian D. Levinson and William B. Raycraft, Chicago, Ill.* J. Pediat. 36: 316–22, March 1950.

A study is presented of 300 cases of diarrhea in patients admitted to the Children's Division of the Cook County Hospital in the five-month period beginning June 1, 1948. These comprised one-sixth of the total admissions with a peak of diarrhea cases in September. The most common symptoms on admission were fever and diarrhea, although in many instances diarrhea did not develop until later. Neurologic manifestations were found in 21% of the cases, and were more common in children over 2 years of age. Neurological symptoms were often so prominent as to mask the underlying disease. In all cases where spinal puncture was performed, the fluid was found to be normal.

In this epidemic, Shigella was found in 28% of the cases, Salmonella in 5%, and Proteus-Paracolon-Pseudomonas organisms in 46%. Shigella was found ten times more frequently in children over one year of age than in those under one year of age. The Proteus-Paracolon-Pseudomonas organisms were found four times as frequently in children under one year of age.

Control studies showed that Proteus organisms in patients under one year and Paracolon in patients over one year of age were of pathogenic significance. Virus studies in 32 cases of diarrhea were negative.

The mortality rate was 8.6%, with a corrected rate, excluding the twenty-four-hour deaths, of 5.3%. This rate compares favorably with that of other series, in spite of the fact that the patients were, with few exceptions, severely ill.

In the therapy of Shigella, sulfadiazine and oral streptomycin were found of about equal efficacy, with a combination of the two as most effective. In diarrhea caused by organisms other than Shigella or Salmonella, the average duration of the diarrhea was cut from approximately five to three days by the use of either sulfadiazine or oral streptomycin. The authors feel that a combination of the early use of intra-

venous fluids, including blood or plasma, the use of sulfadiazine and oral streptomycin, plus good nursing care, will tend to reduce the mortality and hasten the recovery in patients with diarrhea. 17 references. 1 figure. 6 tables.—*Author's abstract.*

Diarrhea in the Otherwise Healthy Infant. *Arthur S. Brackett, Bristol, Conn.* Yale J. Biol. & Med. 22: 429–35, May 1950.

The term "diarrhea" is here defined as the condition arising when at least three watery stools a day occur. There are numerous such cases of unknown etiology in very young infants, which have been called benign and harmless. Their frequency is not generally recognized by the profession. This article suggests that this condition may occur in infants (1) with a colon too small to absorb the contents rapidly enough to supply the body with the necessary water and inorganic salts; (2) with a normal colon into which has been poured too large a bulk of chyme, the feeding being more than necessary for caloric requirements; (3) with a small colon which has to deal with feedings which are excessive in size, i.e., more than necessary for caloric requirements; (4) when there is an ineffective ileocecal sphincter.—*Author's abstract.*

Medical literature seems to have overlooked mention of the optimal size of the individual feeding in the treatment of diarrhea. One factor which contributes to the variability of stool formation in the very young infant is the efficiency of absorption of the water component of the stool.

Studies on the normal frequency of infant stools by the Editor (Major Motility Patterns of the Child's Digestive Tract: Am. J. M. Sc. 207: 782–803, June 1944) gave the following mean values for laxation rate per day in more than 1,000 infants receiving a variety of cow milk formulas:

Age	*1**	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
Mean No. of stools	*2.67*	*2.63*	*2.41*	*2.37*	*2.28*	*2.27*	*2.29*	*2.02*	*1.97*	*1.82*	*1.75*	*1.72*

—Editor.

10. Genitourinary System

The Use of Chloramphenicol ("Chloromycetin") in the Treatment of Infections of the Urinary Tract in Childhood. *J. M. LoPresti, P. Kaufman, B. Olshaker, S. Ross and S. Stevens, Washington, D. C.* Clin. Proc. Children's Hosp. (Washington, D. C.) 6: 177–81, June 1950.

Nine children with urinary tract infections (7 due to *Escherichia coli,*

* Infants aged 3 and 4 weeks only.

1 to *Aerobacter aerogenes*, and 1 to *Serratia marcescens*) were treated with chloramphenicol. The dosage ranged from 75 to 90 mg. per Kg. per twenty-four hours, given orally at three to four-hour intervals. For the younger patients it was necessary to empty the contents of the capsule and dispense it in a suitable vehicle such as syrup of cherry or syrup of Santa Yerba. Disappearance of symptoms and pyuria were noted in all 9 patients. No nausea, vomiting, or diarrhea was encountered during therapy. The duration of treatment ranged from 6 to 14 days, the average being 8 days. In 2 patients, on subsequent urine cultures, it was noted that a relatively resistant strain of *Pseudomonas aeruginosa* developed. One patient with congenital hydroureters relapsed following discontinuation of chloramphenicol. 11 references. 1 table.

11. Growth, Puberty, Adolescence

See Contents for Related Articles

12. History, Biography, Antiquities

See Contents for Related Articles

13. Infectious Diseases, Acute

Coxsackie Viruses and Bornholm Disease. *G. M. Findlay and Elsie M. Howard, London, England.* Brit. M. J. 1: 1233–36, May 27, 1950.

During the course of experimental investigations in a laboratory worker whose serum gave no reaction with Coxsackie viruses, fever and acute intercostal pain developed. Following the attack, complement-fixing antibodies to the Dalldorf 2 strain of Coxsackie virus developed in her blood. In a volunteer inoculated intranasally with the same strain, similar symptoms and positive complement-fixation with Coxsackie virus (Dalldorf 2 strain) developed. The similarity of these symptoms to those of patients suffering from Bornholm disease (epidemic myalgia, epidemic pleurodynia) prompted the examination of sera from patients who had suffered from Bornholm disease. Such cases have given positive complement-fixation with Dalldorf 1 or 2 strains. Patients with nonparalytic poliomyelitis and pains suggestive of coronary thrombosis have given similar positive reactions. One patient with positive complement-fixation to Dalldorf 2 suffered from Bornholm disease in Aden, one with antibodies to Dalldorf 1 in France. A virus resembling Coxsackie virus 2 has been isolated in the London area. The evidence suggests that Coxsackie viruses 1 and 2 are widely distributed in England. 13 references. 2 tables.—*Author's abstract.*

Streptomycin in the Treatment of Influenzal Meningitis. A Study of Ninety Cases, with 96.6 Per Cent Recovery. *Emanuel Applebaum and Jack Nelson, New York City Health Department, New York, N. Y.* J. A. M. A. 143: 715–17, June 24, 1950.

In 90 consecutive cases of meningitis due to Hemophilus influenzae (86 children), streptomycin was given intramuscularly and intrathecally to 73 patients, and intramuscularly only to 17 patients. In addition sulfadiazine was used in all but 1 case, specific rabbit serum in 17, and aureomycin in 1. Of the 90 patients, 87 recovered and 3 died. The 17 patients treated with intramuscular but without intraspinal injections of streptomycin recovered completely. With regard to the adjunctive therapy, it was difficult to evaluate the role of sulfadiazine. There was no evidence that rabbit serum played a contributory role. There were relatively few toxic reactions or neurologic residua. "It is clear from this study that streptomycin is an effective agent in the treatment of meningitis due to Hemophilus influenzae." 1 reference. 4 tables.

Pfeiffer Bacillus Otitis in Young Children (*Småbarnsotiter orsakade av Pfeiffers bacill*). *Gunnar Bjuggren and Gösta Tunewald, Stockholm.* Svenska Läk. tidn. 47: 433–39, Feb. 24, 1950.

In spite of antibiotics and sulfonamide many cases of otitis media remain in children with a prolonged course or of recurrent type, especially in young children under 3 years of age. In 1945, Nielsen reported an incidence of 16% of Pfeiffer bacillus infection in 802 cases of otitis media in children, the majority of whom were under 3 years of age. Of the 6 types of this bacillus, that most frequently encountered was type B. In contradistinction to Pfeiffer bacilli found in normal subjects, the bacillus in the otitis cases was encapsulated. It has been observed that this organism is isolated more frequently from cases of protracted course.

A study is presented of a series of 178 cases in children up to 16 years of age, most of which were fresh clinical cases under treatment with sulfonamides in the Polyclinic of the Eastman Institute. If suppuration persisted after 10 to 14 days, the child was admitted to the hospital. Cases of recurrent otitis media were also included. In this series, the Pfeiffer bacillus was isolated from 15.8% of the cases. In 3.4% the bacilli were not encapsulated and these children yielded a negative complement fixation test. No special lesions due to this bacillus could be demonstrated. In 12.4% of the cases, the bacillus was encapsulated and these cases were almost all in children under 3 years of age.

Of the 22 cases in which the bacillus was found encapsulated, 13, or 7.3% were suffering from primary Pfeiffer bacillus otitis, 6 or 3.4% had otitis with secondary Pfeiffer bacillus infection, and in 3 cases or 1.7% it was doubtful whether the Pfeiffer bacillus infection was primary or secondary. In all except 2 cases the bacillus isolated was of type B.

Pfeiffer bacillus otitis in the beginning is associated with moderate or no fever and the general condition is but slightly affected. The fever then becomes subfebrile and subsides leaving, principally, local symptoms. There is a heavy discharge and usually a coincident rhinitis with very thick mucus. In one case of conjunctivitis this bacillus was isolated from the secretion. Under intensive sulfonamide and penicillin therapy, the otitis usually heals but recurrence is common, and infection of the nasal cavities may persist long after the otitis has disappeared. Constant recurrence of this type of otitis may eventually affect hearing. Attention is drawn to the probable relation of Pfeiffer bacillus otitis media to Pfeiffer bacillus meningitis. 12 references. 1 figure. 1 table.

Evolution of Neurological Signs of Early Anterior Poliomyelitis. *Lewis J. Pollock, Benjamin Boshes, Isidore Finkelman, Frederick Hiller, Herman Chor, Meyer Brown, Alex J. Arieff, Erich Liebert, Eli L. Tigay, Maurice Schiller and Irving C. Sherman, Northwestern University Medical School, Chicago, Ill.* Am. J. Dis. Child. 79: 973–87, June 1950.

Eleven neurologists were rotated on tours of duty of 6 hours each day and night for 30 days on the poliomyelitis ward of the Chicago Municipal Contagious Hospital. Frequent regular examinations were made of each patient admitted with the suspicion of poliomyelitis. Excluding those who died or became respirator cases, 30 patients were studied over sufficiently long periods to permit an analysis. They were divided retrospectively into two groups—Group A (16 patients) in whom severe paralysis developed, and Group B (14 patients) who had unparalyzed or slightly weakened muscles.

No instance of muscle spasm—defined as a "sustained reversible tetanic contraction"—was encountered while paralysis was developing. Tenderness and stretch pain, often associated with spasm and believed by some to be manifestations of a spastic state, were infrequent and found only after paralysis had occurred.

Neck rigidity occurred in 92.4% of examinations, although usually it was possible to move the head forward to some degree without pain. Brudzinski's sign was discovered in 37%, but not as pronounced as in meningitis. Kernig's sign was found in 39%, but the leg could generally be extended on the thigh to a greater degree than in meningitis. The frequency of Kernig's sign increased as the disease progressed (17% before 100 hours; 54% after). The authors suggest that this and other signs (including neck rigidity) may signify an unconscious effort at protective splinting, the pain effectively limiting the range of motion or maintaining a position so as not to stimulate the cord membranes and nerve roots.

Severe paralysis was observed as early as 18 hours after onset. For most of the cases in which severe paralysis developed the peak did not occur until after the 75th hour. Weakness did not always progress steadily to paralysis. Sometimes a muscle would repeatedly regain strength to a point nearly normal, and then become weak again. This variability from hour to hour was striking. Weakness sometimes remained stationary for a considerable number of hours and then went on to either paralysis or remission.

The deep (stretch) reflexes were absent in 91% of muscles destined to become paralyzed. The earlier the reflexes disappeared, the more severe the paralysis. When the reflexes disappeared after 125 hours severe paralysis did not occur. Reflex changes thus appear to be of value in prognosis.

There was an early loss of abdominal reflexes in cases in which severe leg paralysis developed later. In these cases, the abdominal reflexes were absent in 85% of the examinations up to the 100th hour after onset of disease, whereas in those who failed to develop lower limb paralysis, only 3.2% lost these reflexes. The loss of abdominal reflexes can be considered a sensitive index of possible leg involvement.

The plantar reflexes (contraction of toes when the sole is stimulated) were absent at various times in 35% of the examinations of those with severely paralyzed legs, and in only 2.3% of those with moderate weakness in the legs. Babinski's sign was found rarely and inconsistently in these cases.

Irritability of the muscles to direct mechanical or electrical stimulation was increased in some of the paralyzed muscles, especially in the arms. There was no correlation between the degree of paralysis and increased muscular irritability. It was found more often earlier in the disease than later. There was some response to direct electrical stimulation in all muscles, even when clinically completely paralyzed. 9 references. 9 charts.

Electrophrenic Respiration in Acute Bulbar Poliomyelitis: Its Use in Management of Respiratory Irregularities. *Stanley J. Sarnoff, James V. Maloney, L. Charlotte Sarnoff, Benjamin G. Ferris and James L. Whittenberger, Harvard School of Public Health, Boston, Mass.* J. A. M. A. 143: 1383–89, Aug. 19, 1950.

Dysfunction of the respiratory center in the medulla in bulbar poliomyelitis usually results in irregular rhythm and depth of breathing as well as in lack of coordination of various respiratory muscles. The breathing muscles may be structurally intact, but death can result from inadequate aeration from such inefficient breathing. The mechanical respirator is usually ineffective in this disturbance because of the difficulty of synchronizing the respirator's rate with the irregular arrhythmic breathing produced by the damaged center.

Artificial respiration by electric stimulation of one of the phrenic nerves (EPR) has been proven experimentally to work excellently when dealing with respiratory emergencies in which a relatively short period of artificial respiration is required. It was found that EPR immediately took over the control of breathing, abolishing in a few seconds all efforts at spontaneous respiration. For this reason it seemed possible that EPR in bulbar poliomyelitis could command the respirations and give the patient controlled, regular breathing in place of the spasmodic, incoordinated type resulting from the virus-damaged center.

EPR was tried in 9 patients with bulbar poliomyelitis in the summer of 1949. The intensity of involvement in the cases differed considerably, but all had respiratory irregularities of central origin. EPR overcame the irregularities in each case and provided effective ventilation for varying periods of time.

From physiologic studies carried out before, during and after EPR, conclusions were drawn regarding the efficacy of EPR in bulbar poliomyelitis. Respiratory center involvement does contribute significantly to the symptoms. Each of the 9 patients had respiratory irregularities in the absence of (or independently of) airway blockage. This confirms other observations that positive correlation exists between lesions in a certain area of the medulla (i.e., the respiratory center) found at autopsy and clinical histories characteristic of respiratory irregularities noted during illness.

In addition to irregularities of rhythm and incoordination of muscular breathing effort, respiratory center involvement may give rise to another type of difficulty called "hypoventilation syndrome." When uncomplicated, this state is characterized as under-breathing—shallow and slow respiration resulting in hypoventilation. Hypoventilation is attributed to the lowered sensitivity of the respiratory center to a high CO_2 tension in the blood. The center fails to send sufficient stimuli to the respiratory muscles to produce normal breathing.

It seemed clear from some of the case histories that a marked disorganization of the respiratory center may be followed by a complete recovery if the demands of the respiratory and circulatory systems are met during the critical period. It is therefore most important to tide the patient over the crucial few days following the onset, while waiting for recovery of the autonomic center.

It was possible to overcome respiratory irregularities in the milder cases by using high pressure gradients (i.e., large negative pressure) in a tank respirator. But the authors believe the procedure to be undesirable because this type of respiration has adverse effects on circulation, lowering the cardiac output and thus further diminishing oxygen supply to tissues. Higher pressure gradients to break through the respiratory incoordination of these cases is not without danger when the circulatory status is already below normal. EPR does not employ positive or nega-

tive pressure and does not depress cardiac output or circulatory tone.

How much ventilation to give a patient at any particular time is difficult to answer. Even in a seemingly well-ventilated patient with a satisfactory oxygen content, the CO_2 excretion at the alveoli of the lungs may be impaired. An analyzer for either blood or alveolar CO_2 is needed, attached to the EPR machine in such a way that the changes in the gas tension, as registered in the analyzer, will automatically regulate the voltage of the machine and the amount of stimulation given to the phrenic nerve. This will endow the electrophrenic respirator with some of the autonomic qualities characteristic of the respiratory center.

Whenever one phrenic nerve was in working order, EPR suppressed spontaneous respiration. This enabled it to abolish the arrhythmic and incoordinated breathing due to respiratory center disorder. EPR was then maintained until the patient no longer required artificial respiration or his breathing was coordinated to the rhythm of a tank respirator. One eventual application of the electrophrenic respirator may be to adapt the refractory patient to the tank respirator.

Unlike positive pressure breathing or that given by the tank respirator, EPR was found to encourage venous return to the heart, and maintain higher cardiac output. This is highly desirable, since maintaining the circulatory status of these patients is important in warding off the ever-present menace of anoxia. Pathologically, elevated blood pressure was repeatedly reduced in some of the patients. A rapidly shifting blood pressure, sometimes reaching very high levels, is not an uncommon finding in severe bulbar cases and may represent an involvement of the vasomotor center in the medulla or fluctuations in the adequacy of ventilation.

EPR counteracted restlessness in several patients, thereby facilitating the handling of these patients. Nursing care was also easier than in a body-enclosing respirator because of fewer mechanical obstacles.

So far no evidence has been found of vagal overstimulation due to EPR. Slowing of the heart rate, the cardinal sign of an over-active vagus, was not noted in this group nor in the 100 normal volunteers in whom the EPR was first tried.

This new and promising approach to the management of bulbar poliomyelitis is still under exploration. The apparatus requires an experienced technical supervisor who must be in constant attendance while it is operating. Further reports of additional experiences must be awaited. —EDITOR.

Epidemiologic Studies of Q Fever in Southern California. *Joseph A. Bell, M. Dorothy Beck and Robert J. Huebner, Bethesda, Md* J. A. M. A. 142: 868–72. March 25, 1950.

This report summarizes the important findings resolved from many specific epidemiologic studies embracing some 10,000 people of selected

population groups in Los Angeles. The main purpose of these studies was to determine the chief sources of human infection. The complement fixation test performed by a particular technic seemed a remarkably satisfactory index of recent human infection with Coxiella burnetii. By using this test on a large sample of the general population it was estimated that more than 50,000 persons in this area have been infected with C. burnetii during recent years. Many of these infections had produced an acute febrile illness which was not previously recognized as Q fever. This establishes that Q fever can be endemic and can persist unrecognized for years as a sizable medical problem in a large community of high social and economic level.

The most frequent and by far the most important sources of human infection were local dairy cows, their very young calves and some of their raw products, particularly raw milk and hides. The persons most likely to have been infected were those who had used raw milk in their households, those whose residence had been located near a dairy or livestock yard, those who had worked in industries handling live or recently killed local dairy cows and young calves (e.g., employees on dairies, in meat-packing plants and fat-rendering plants), and employees handling the raw products of such animals, e.g., employees of creameries and hide plants. This experience further confirms that domestic livestock is an important source of human infection. It shows that the environment and raw products, particularly hides and milk, of infected dairy cows are important sources of human infection and disease. 6 references. 7 tables.—*Author's abstract.*

Penicillin Therapy in Scarlet Fever (*Behandeling van roodbonk met penicilline*). *J. E. Minkenhof, H. Peeters and M. C. Visser, Amsterdam, The Netherlands.* Nederl. tijdschr. v. geneesk. 93: 3877–84, Nov. 12, 1949.

Four hundred and seventy-two patients with scarlet fever were treated with 100,000 to 200,000 units per day of penicillin for six days. Complications were three times more frequent in the control groups than in those treated with penicillin. Two of the patients who were treated with penicillin had a second attack of scarlet fever after intervals of three and five months. Two relapses occurred. In 131 cases followed by throat swabs (in the majority of those treated) cultures for hemolytic streptococci became negative with penicillin therapy. Some, however, remained contagious. 37 references.—*R. H. Stoppelman.*

Tetanus Neonatorum (*Tetanus neonatorum*). *J. H. de Haas, Leiden, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 164–72, June 1949.

Two cases of tetanus neonatorum are described which were traced to contamination from the umbilical scissors. The incubation period was

seven days in the successfully treated case and four days in the fatal one. Phenobarbital and penicillin were given in high dosage. Both were administered orally to avoid the convulsant effect of repeated injections. Serum is not as effective as it was once thought to be. The complication of spondylolisthesis was encountered; the second lumbar segment was displaced posteriorly, but later became corrected spontaneously. Recent literature suggests an increase of tetanus neonatorum in the Netherlands. 23 references. 2 figures.—*H. Bartels.*

14. Infectious Diseases, Chronic

Reactivation of Rheumatic Fever by Smallpox Vaccination. *Paul Freud, George D. Rook and Andrew Brunhofer, New York, N. Y.* J. Pediat. 36: 635–40, May 1950.

Six cases of reactivation of rheumatic fever following smallpox vaccination are described. All had inactive rheumatic fever at the time of vaccination. Five other inactive rheumatic fever patients had no flareup following vaccination. The unpredictability of rheumatic fever recurrences is discussed. It is suggested that rheumatic children should only be vaccinated if exposed to smallpox. 10 references.

Three Cases of Transplacental Tuberculous Infection of Infants (*Sur trois cas de tuberculose transplacentaire du nourrisson*). *N. Martrou and P. Martrou, Limoges, France.* Nourrisson 38: 79–84, March–April 1950.

Transplacental tuberculous infection of infants is rare. Three cases are reported in infants delivered of mothers who had advanced pulmonary tuberculosis with cavitation in whom tuberculosis developed in the first few weeks of life. In all these cases no evidence of involvement of any other organ except the lungs was found in the mother. The infant, in each case, was kept from contact with the mother from the time of delivery. The first infant was premature (7 months); he did not gain weight normally and early in the second month of life fever developed and he died before the end of the month. Autopsy showed miliary tuberculosis of the lungs. The second infant gained weight normally until the sixth week of life, then after a time began to lose weight and died in a state of cachexia at 5 months of age. The chief symptoms were slight fever and an unusual pallor. At autopsy the right lung showed caseation with one small cavity and the left lung showed miliary tuberculosis; there were also miliary lesions in the spleen and in the renal cortex. The third infant gained weight normally until the sixth week; following that, fever, pallor and some enlargement of the liver developed. As the x-rays showed pulmonary lesions, treatment with streptomycin was begun; the doses for the first two months was 0.30 Gm. daily in divided doses. Since there was no definite improvement, two months later the dose was increased to 0.60 Gm. daily in three divided doses. Calcium and iron were also given, this resulted in stabilizing the

temperature but there was no definite symptomatic improvement for three months. The appetite then improved; there was a regular gain in weight and x-rays showed regression of the pulmonary lesions. When the patient was discharged from the hospital at the age of 9½ months, there was no sign of activity of the disease. The general condition has since improved.

All infants born of mothers with advanced tuberculosis should be carefully followed up, with repeated tuberculin tests (which were positive in the 3 cases reported), and x-ray examination of the lungs every fifteen days for the first three or four months. 6 references.

Treatment of Primary Pulmonary Tuberculosis with Para-Amino-Salicylic Acid and the Sodium Salt (*La terapia con acido para-amino salicilio ed il suo sale sodico in alcune forme di tbc. pulmonare infantile*). *U. Pelligrini and G. Maini, University of Parma, Italy.* Il Lattante 21: 1–41, Jan. 1950.

Twenty children aged 1 to 10 years with pulmonary tuberculosis were treated with para-aminosalicylic acid and its sodium salt. The dose was 0.4–0.5 Gm. per Kg. per day, given in courses of 20 days for 3 to 4 such courses. The drug was well tolerated by the children. The single complication was "black-tongue" in 3.

An early improvement in the general status of the patient occurred, with improved appetite, weight gain and diminution in fever. The latter occurred within 3 to 8 days of initiation of therapy even when previous therapy with streptomycin had failed. Fever also fell rapidly in 2 patients with miliary and meningeal tuberculosis. Some had slow radiologic improvement. 21 figures. 1 table.—*A. M. Bongiovanni.*

Streptomycin in Tuberculous Meningitis. Report of the Ministry of Health. Lancet 2: 230–31, Aug. 1950.

A review was made of 369 bacteriologically proven cases of tuberculous meningitis in order to evaluate the efficacy of streptomycin in its treatment. During an observation period of 10 to 22 months the overall mortality rate was 71.4%. The 28.6% survivors (104 cases) were followed for a longer period, varying from 22 to 33 months. At that time all were still alive, 84.6% of them (88 patients) were clinically well and 57% (59 patients) had normal cerebrospinal fluid. No relapse occurred during this period in any patient whose spinal fluid became normal. Standards for normal spinal fluid included not more than four cells per cu. mm., protein not more than 40 mg. per 100 ml., and sugar not less than 45 mg. per ml.

The institution of treatment early in the course of the disease was found to be very important. There were 48% survivors after 30 months of followup in those treated "early", whereas when treatment was begun in an "advanced" stage of the disease there were only 12% survivals after 13 months, and 10% after 30 months. Patients treated with intra-

muscular streptomycin alone rarely responded satisfactorily. An intensive prolonged intrathecal course of streptomycin in conjunction with its intramuscular use gave the best results. When the total number of daily intrathecal doses was less than 50% of the total number of days of intramuscular therapy, results were considerably poorer than when the daily intrathecal streptomycin was given for a least two-thirds of the total number of days of intramuscular therapy.—*W. C. Blair.*

Streptomycin Therapy in Tuberculous Meningitis (*Behandeling van meningitis tuberculosa met streptomycine*). *S. van Creveld and M. R. H. Stoppelman, Amsterdam, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 77–92, 1949.

Experiences with streptomycin in 7 children with tuberculous meningitis are described. Their ages varied from 15 months to 6 years. Five were admitted in the late phases of the disease. Three patients had coincident miliary tuberculosis of the lungs. In all instances tubercle bacilli were isolated from the spinal fluid. One case had no changes in the lungs at autopsy, but one mesenteric node was enlarged. From this node as well as from the spinal fluid, tubercle bacilli of the bovine type were cultured. In all the other cases the organisms were of the human type.

Three children recovered, 2 with severe mental and neurologic residues. The other is still in excellent condition one and a half years after therapy was started. In 5 children 45 mg. streptomycin per Kg. body weight daily were injected intramuscularly and 25 to 100 mg. intrathecally. One child was treated with streptomycin and promine. The patient who recovered without defects received 45 mg. streptomycin per Kg. daily during the first ten days and then 90 mg. per Kg. daily over a period of five months. 10 references. 1 figure.—*M. R. H. Stoppelman.*

Effect of TB I 698 and TB IV on the Course of Tuberculous Meningitis (*Der Einfluss von TBI 698 und TB IV auf den Verlauf der tuberkulösen Meningitis*). *Karl Ludwig Radenbach.* Beitr. z. Klin. d. Tuberk. 102: 377–90, Heft 3, 1949.

Following a short review of the basis for treatment of tuberculous meningitis with TBI 698 and TB IV (Domagk), the author reports 5 patients with tuberculous meningitis treated with TBI 698 or a combination of the two synthetic preparations. Although the outcome was fatal in all 5 cases, a favorable effect of the treatment was unmistakable. The combined local application of these two drugs had a favorable effect not observed following the use of TBI 698 alone. The combined treatment brought about short or long remissions, with an improvement in symptoms, and reduction of the cell count in the cerebrospinal fluid, a drop in temperature and in some instances prolonged survival. Several writers have drawn attention to this prolonged course obtained by the use of TBI 698. According to Heelmeyer, TBI 698 has little effect in tuber-

culous meningitis. He emphasizes the nonspecific effect of TBI 698 in hypergic and allergic processes.

Comparing his results with those reported in the literature in regard to results of PAS therapy in tuberculous meningitis, he found that the results of this combined TBI 698 and TB IV therapy are at least equally good. One advantage, as compared with the less easily obtained streptomycin, would seem to be that even following prolonged administration, no bacterial resistance to the drug is developed. It is suggested that this treatment be used in combination with streptomycin therapy, which does not seem to solve the problem. The tuberculostatic effect of TBI 698 and TB IV is superior to that of other drugs (penicillin, sulfadiazine, etc.) suggested for combined treatment with streptomycin.

In 1948, Gehrt reported a case of tuberculous meningitis in an infant 2 years of age, in which lasting cure followed administration of TBI 698. Several patients with miliary tuberculosis cured by this treatment have been reported.

Recently, Domagk (1949) described a water-soluble thiosemicarbazide preparation, TB VI, for intravenous and subarachnoid administration. With combined treatment of TBI 698 and TB VI (0.4 Gm. TBI 698 daily and 0.02 to 0.03 Gm. and more of TB VI diluted with cerebrospinal fluid to 5 to 10 cc. by subarachnoid injection), a patient 64 years of age with diagnostically confirmed tuberculous meningitis was symptom-free after three and one half months, although moribund on admission. TBI 698 is now known commercially as "conteben." A combined TB VI and streptomycin treatment is recommended for tuberculous meningitis. 60 references.

Problems of the Toxic Effects of Thiosemicarbazone in the Treatment of Tuberculosis in Children. (*Probleme der Thiosemicarbazongiftwirkung bei der Behandlung kindlicher Tuberkulose*). *Fritz Heepe, Göttingen, Germany.* Mschr. f. Kinderh. 97: 349–56, Heft 8, 1949.

The toxic effects of thiosemicarbazone (TBI) may vary from slight disturbances to fatal effects. Among the undesirable effects may be malaise, anorexia, vague gastric distress, heartburn, eructations, taste anomalies, ravenous hunger, a craving for acids and disgust for sweets, nausea, vomiting, constipation, palpitation, arrhythmia, headache, vertigo, as well as ocular pain, photophobia, excessive perspiration, urticaria, a hemorrhagic tendency and excessive menstrual bleeding. In severe cases there may be loss of weight, jaundice, enlargement of the liver, somnolence, coma or convulsions. There may be changes in the blood and urine. Individual sensitivity varies greatly. In some instances death may ensue in three days, with symptoms of involvement of the central nervous system. Autopsy reveals toxic fatty degeneration of the liver, edema, degeneration of the brain and other degenera-

tive lesions. Slight toxic symptoms usually subside following temporary interruption of treatment, or reduction in dosage.

In the year 1948 to 1949, 32 children were treated at Göttingen with thiosemicarbazone. Almost all these cases were tuberculosis (primary or secondary infiltration, miliary tuberculosis and tuberculous meningitis). Some cases of lupus and tuberculous osteomyelitis were included. The duration of treatment was 2 to 4 months. The daily dose was 2.5 to 25.0 mg. per Kg. and the total dose was up to 800 mg. per Kg. The dose was diminished immediately if any untoward symptoms appeared. More than one-half of the patients had transitory symptoms. Severe intoxication developed in only 3 cases, one of which proved fatal. Severe intoxication may develop suddenly, without warning signs of disturbance in liver function or other signs of toxic effect.

Attention is directed to intercurrent infection during TBI therapy. This may be the cause of collapse of liver function already impaired by the drug.

With all precautions as to dosage and careful observation for signs of intoxication, TBI may prove a life-saving measure in tuberculous children. The symptoms of tuberculous meningitis may resemble those of TBI intoxication. The optimal dose for children is 0.25 to 0.375 Gm. daily. Severe toxic symptoms may be expected following doses of 15 mg. per Kg. and up. Sensitive patients may have ill effects from even smaller doses. It is best to attain the maximal dose by cautious gradual increase over a period of four to six weeks. 55 references. 1 table.

The Incidence of Epidemic Meningitis in Children Suffering from Tuberculous Meningitis (*Das Auftreten von epidemischer Meningitis bei an Meningitis tuberculosa leidenden Kindern*). *K. B. Choremis, N. Zervos, B. Konstandinidis, S. Pantazis and G. Belissaropoulus, Athens, Greece.* Schweiz. med. Wschr. 80: 138–39, Feb. 11, 1950.

The unfavorable effect of infectious diseases complicating tuberculous meningitis in children is stressed. The authors had never observed epidemic meningitis as a complication of tuberculous meningitis before the advent of streptomycin, but have recently observed 6 cases, 4 of which are described in detail. The onset of the epidemic disease in children with tuberculous meningitis is marked by a sudden severe exacerbation of symptoms, with marked elevation of temperature in cases in which fever had hitherto been absent or low, and an exacerbation of the meningeal symptoms. Such a change is, of course, more obvious in cases in which the symptoms of the tuberculous meningitis were not very severe at the time. Spinal puncture reveals an opaque spinal fluid under increased pressure, with an increase in albumin and a high cell count. Meningococci can be demonstrated in the culture and also microscopically. Diagnosis is simple, and difficulties may be encountered only if the tuberculous meningitis is in an

acute stage at the time of the secondary infection. The prognosis for the double infection is extremely grave, and depends upon the state of the primary infection and prompt diagnosis of the complication. Intensive treatment of the epidemic meningitis is indicated. Intraspinal injections of streptomycin should be replaced by penicillin injections and intramuscular injections of streptomycin should be continued with supportive sulfonamide therapy. Three of the 6 cases proved fatal. In 2 of these, death was definitely due to the epidemic meningitis. Since epidemic meningitis is not considered very contagious, precautions for isolation have not been too strict heretofore, but will be observed in the future since it is believed that children with tuberculous meningitis may be particularly susceptible to this infection, owing to the pathologic changes already present in the meninges and diminished resistance. Five of the 6 patients were under 3 years of age. 3 references.

15. Liver, Kidneys and Spleen

Aspiration Biopsy of the Liver in Children? (*Aspiratie lieverbiopsie bij kinderen?*). *C. van Beek and A. J. Ch. Haex, Leiden, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 11, April, 1949.

The authors performed about 1,500 needle biopsies of the liver in adults without casualty. They consider the fixation of respiration in children a great risk, and advise against aspiration biopsy of the liver. 8 references.—*F. Kuipers.*

A Contribution to the Knowledge of Banti's Syndrome. With Special Emphasis on Differential Diagnosis and Treatment. *L. E. Carlgren, A. Lichtenstein, and Ph. Sandblom, Stockholm, Sweden.* Acta. pædiat, Supp. 75: 10–56, 1949.

All attempts to get rid of the term "Banti's syndrome" have hitherto been futile. It has not been possible to find a better name for all those disease complexes of different or unknown etiology, with or without liver damage and with or without hemorrhages of the digestive tract, in which there occurs considerable, usually progressive hypertrophy of the spleen with the character of fibro-adenia or congestion. The pathogenesis of these conditions is still obscure and subject to widely divergent opinions. Whatever the point of view adopted it would seem most logical to classify the cases into (1) those in which the liver is chiefly affected (splenomegalous hepatic cirrhosis); (2) those in which the damage seems to have affected particularly the portal vein and its tributaries (so-called portal or splenic vein thrombosis); and (3) those in which for the most part the spleen alone is the seat of the pathologic changes (so-called idiopathic splenomegaly).

Of 14 patients treated at the Kronprinsessan Lovisa's Children's Hospital in Stockholm, 7 belonged to the group splenomegalous hepatic

cirrhosis and 7 to the group with established or probable obstruction of the portal vein. The former showed clinical signs of liver disease (icterus, urobilinuria), and these signs were lacking in the cases belonging to the second group. Even in some of the latter, however, liver function tests turned out positive to a slight extent. In the cases with established or probable portal obstruction, bleeding from esophageal varices was an early and constant sign, whereas it was infrequent and late in splenomegalous hepatic cirrhosis. Ascites occurred in both groups; while transient and without prognostic significance in the cases of portal vein disease, it was always an ominous sign in liver cirrhosis. The prothrombin time was often prolonged in the cirrhosis cases, never in the other group. Blood examination did not yield much of value in differential diagnosis, with one exception: the sedimentation rate was invariably increased in the cirrhosis cases, especially in the earlier stages; in all cases in the other group, except one, it remained normal.

In one of the cirrhosis cases, splenectomy was done on a faulty diagnosis of Werlhof's disease; in the remainder treatment was conservative. Of this group 4 are still alive five to nine years after beginning of treatment. In all the cases with established or probable portal obstruction splenectomy was performed, in 6 cases as an isolated measure. The results were extremely discouraging: bleeding recurred in all within two years after operation. In 3 patients, in addition, an anastomosis between the portal and caval system was made. In 2 of the patients splenectomy was previously done and even in the third case the anatomical conditions were unfavorable so that it was impossible to get a satisfactory shunt. All 3 continued to bleed after operation. In spite of the poor results obtained in these cases it is felt that splenectomy should not be undertaken in cases with Banti's syndrome without planning an anastomosis operation. Recent reports from the American literature indicate that good results may be expected in many cases with end-to-side splenorenal anastomosis.

Two of these patients have died since this paper was published. In 3 additional cases, however, with established portal obstruction treated with splenectomy and splenorenal anastomosis of the suture type, the operation seems to promise a good function of the shunt. 33 references. 2 tables.—*Authors' abstract.*

Prognosis of Chronic Hepatitis in Children. *George B. Logan, Mayo Clinic, Rochester, Minn.* Proc. Staff Meet., Mayo Clin. 25: 299–304, May 24, 1950.

Seventy-four children who had chronic disease of the liver were studied. Sufficient follow-up data were available to permit comment as to the condition of 53 of these children. Forty-one, or approximately three fourths of the 53, died within 5 years after the onset of the disease. One patient, however, lived for 23 years after the onset. Seven patients were still living 6 to 21 years after the onset of the disease.

The impression has been gained, from observation of some of these children, that chronic hepatitis may be a reversible process. Before a definite conclusion can be drawn in this respect, however, a longer period of observation will be necessary. In some instances, what have been considered reversible processes may, instead, represent instances of long latent periods of the disease. 3 references. 1 figure. 3 tables. —*Author's abstract.*

Chronic Acidosis of Renal Origin in Infancy. *Weston M. Kelsey, John B. Reinhart and John Fishel, Bowman Gray School of Medicine of Wake Forest College, and the North Carolina Baptist Hospital, Winston-Salem, N. C.* Pediatrics 5: 689–94, April 1950.

Two infants are described who had anorexia, vomiting, constipation and failure to gain weight, followed by sudden onset of fever, signs of acidosis, and collapse. Physical findings were minimal. The essential acid base disturbances were increased serum chloride and decreased plasma bicarbonate. In spite of the acidosis both excreted a persistently alkaline urine. Except for suggestive evidence of nephrogenic diabetes insipidus in one there was no evidence of any other tubular defect. Both were improved dramatically by the administration of alkaline salts.

It was concluded that these two infants who had unexplained signs of anorexia, vomiting, dehydration, fever and acidosis probably had defective reabsorption of bicarbonate in the renal tubules. One, after a year of therapy, has recovered and developed normally without further treatment. The other still requires alkaline salts to prevent the development of acidosis. 16 references. 2 tables.

Clinical Recovery Following Prolonged Anuria in an Infant Two Months of Age. Report of a Case of Potassium Bromate Intoxication. *S. A. Warren and W. V. Gross, Jr., Sinai Hospital, Baltimore, Md.* Pediatrics 5: 954–58, June 1950.

This is the report of success in treating an infant with acute renal insufficiency ("lower nephron nephrosis") caused by potassium bromate poisoning. This 2-month-old infant was inadvertently fed 42 Gm. of a water-clear solution containing potassium bromate in concentration of 2.5 Gm. %, employed to set waves in the hair. Two hours later the baby had a liquid stool. After that he vomited most of his feedings and became pale and listless.

The error was discovered 17 hours after the poison was ingested. When hospitalized at that time he was moderately dehydrated. He was given 90 cc. of 5% glucose solution by mouth and 80 cc. of 10% gluco e intravenously. No urine was passed after the first day until 4½ days later, the anuria being of 107 hours' total duration.

On the first hospital day the infant had frequent tonic and clonic

convulsions despite phenobarbital sedation. He was given 60 cc. M/6 Na lactate by hypodermoclysis, and 90 cc. of 5% glucose solution by mouth was fed every 3 hours. Blood pressure and heart rate were good. There was a leukocytosis of 18,300. Serum Na was 133 mEq./l. and serum K 5.6 mEq./l. (Normals in this laboratory were 140–146 for Na and 4.5–5.0 for K.). Serum Ca 8.3 mg./100 cc.; serum P 8.8 mg./100 cc.; blood urea 136 mg./100 cc.; Cl 95 mEq./l.; CO_2 combining power 31 vol./100 cc. Beginning on the second hospital day, fluids were not given for 24 hours, because in the preceding 12 hours the patient had received a total of 760 cc. by all routes. He was restricted thereafter to approximately 120 cc. each day, the estimated insensible fluid loss. The fluid was given by mouth as 20% glucose solution. When urine secretion began, additional fluid was given, equivalent to the day's urine volume. During the anuric period the patient was drowsy but took feedings well. He had 5 to 8 green semiliquid stools each day. Blood urea rose steadily to 296 mg./100 cc. the day he voided and continued to rise, reaching a peak of 360 mg./100 cc. on the fourteenth day after ingestion of potassium bromate. Urinary output was 23 cc. the first day of voiding and on the following days was 22, 47, 43, 62, 45, 60 and 50 cc. After 10 weeks of hospitalization, the average daily urinary output was about 200 cc. There was no period of profuse diuresis.

Nineteen days after admission the blood urea was 128 mg./100 cc. and the urinary output was 110 cc. A formula of evaporated milk and 20% glucose was given. During the following 2-week period this feeding was discontinued several times because of vomiting and abnormally high serum Na and K.

The urine specimens after the period of anuria contained a trace of albumin, a few leukocytes and much granular debris. The initial specific gravity was 1.006. Ten weeks later the specific gravity was between 1.008 and 1.009, and the blood urea was 78 mg./100 cc.

Tracheobronchitis developed and the patient was acutely ill from the twenty-first to the twenty-seventh hospital days. He responded well to steam inhalations, penicillin and aureomycin. During the first five hospital days, the daily serum potassium values were 5.6, 6.5, 7.1, 5.6 and 5.6 mEq./l. The initial elevation was probably due to ingested potassium bromate. The progressive rise was due in part to the inability of the injured kidneys to excrete this cation and also to increased stores liberated by endogenous protein catabolism. Repeated electrocardiograms in the first three days demonstrated high peaked T waves in leads II and CF_4, characteristic of hyperpotassemia.

Blood urea was checked at approximately monthly intervals. Except for one reading of 234 mg./100 cc. eight months after admission, this varied between 82 and 124 mg./100 cc. Serum sodium values were repeatedly normal. Serum potassium was elevated to 7 mEq./l. for 8 months, and then fell. Urine dilution test at 8 months revealed a fixed specific gravity at 1.013–1.011. The phenolsulfonphthalein excretion

was 40% in two hours. Clinically the patient seemed well. 13 references. 1 table.

This paper illustrates that recovery from anuria of several days' duration is possible in infants. The physiologic basis of therapy is the maintenance of body water and electrolyte as nearly normal as possible and is applicable to marked impairment of renal function in acute nephritis, so-called lower nephron nephrosis and to a less extent, chronic glomerular nephritis. Success apparently depends on the fact that some types of renal disease lead to injury of tubular cells which make them practically indifferent to water and electrolyte and hence practically all the glomerular filtrate is reabsorbed. The tubular cells often recover sufficiently to permit survival if anuria does not persist longer than 10 to 14 days. Death may develop owing to a rise in the extracellular concentration of potassium, to changes in body water and electrolyte, or certain toxic effects of protein metabolism when renal function is poor. However, circulatory failure aggravates these effects whether due to changes in body water and electrolyte or a rise in blood pressure. All these factors can be favorably modified or postponed by proper fluid therapy.

The opinion that oliguria responds to high intakes of water and electrolyte is based on the fact that oliguria or anuria may be produced by marked losses of water and extracellular electrolyte or shock. If water and electrolyte are restored and shock overcome, this type of oliguria is readily corrected since it is primarily circulatory. However, the anoxia accompanying shock and dehydration may damage the tubular cells and lead to the type of anuria which responds slowly under consideration to therapy directed towards maintenance of normal body water and electrolyte.

The chief functions that are impaired are: (1) excretion of water, (2) excretion of electrolyte, and (3) excretion of urea and other waste products. Little can be done about the rise in nonprotein nitrogen by fluid therapy alone. However, the rate of breakdown of tissue protein can be diminished by providing sufficient glucose to induce maximal protein sparing. About 5 Gm. of glucose per 100 calories metabolized achieves this end and more, does not diminish protein metabolized although it decreases the metabolism of fat. The latter is probably not important since treatment must succeed within about 10 days in restoring renal function. The rise in blood urea is not the cause of death since urea is not toxic.

Administration of too much water will dilute body fluids and may produce water intoxication and the impairment of circulation accompanying low serum electrolyte concentrations. Defective excretion of electrolyte produces disturbances in acid-base equilibrium—particularly acidosis. For this reason sodium lactate should be combined with sodium chloride when correcting deficits of extracellular electrolyte. If too much sodium chloride is given some electrolyte concentrations in-

crease. If both water and sodium salts are given in proportion edema is produced. The metabolism of tissue protein frees potassium which may lead to sufficiently high serum concentrations to produce potassium intoxication. For this reason solutions containing potassium are contraindicated during oliguria. Losses of electrolyte in diarrhea stools or vomitus should be replaced and acidosis should be corrected by appropriate doses of sodium lactate and sodium chloride.

Except for vomitus and stools, water expenditure is confined to losses from the skin and lungs which will be about 50 cc. per 100 calories metabolized if there is no sweating. Slightly more than this amount may be given but the water balance should be checked by weighing the patient daily. The weight should be kept fairly constant although a slight loss of tissue is to be expected. Electrolyte concentrations and acid-base equilibrium should be checked daily by daily serum analyses. The skin losses of sodium chloride are small if there is no sweating—about 1.5 mm. per 100 calories metabolized (80 mg. NaCl). Except when there are losses in loose stools or vomitus, the salt intake should be but little above this amount if there is no sweating. Retention of phosphate may lead to lowering of serum calcium which requires calcium therapy.

As long as there is no vomiting, fluids may be given orally. Food (chiefly sugar) may be added which contains no potassium or protein. Fruit juices contain considerable potassium. The metabolism of protein leads to toxic reactions and hastens death in experimental animals and patients. Amino acids and proteins are contraindicated.

Recovery from the several aspects of renal function is not simultaneous. Therapy is modified accordingly. Water intake is increased as urine is excreted. Sometimes water diuresis is accompanied by excessive losses of sodium chloride while in other cases water diuresis may occur with little loss of salt. When water is excreted it is desirable to promote large urine volumes (80 to 100 cc. per 100 calories metabolized) by large water intakes. Salt intake is governed by testing the urine for chloride and the serum for electrolyte concentrations. During diuresis, potassium may be excreted excessively but if food is taken it is seldom necessary to give additional potassium.

Antibiotic therapy is usually indicated but the amounts given should not be greater than one third to one half the usual dose. Sulfonamides are probably undesirable unless pyelitis is present but even in pyelitis, antibiotics are probably more effective as well as safer.—D. C. D.

16. Metabolic and Systemic Disorders

Degenerative Changes in Young Diabetic Patients in Relationship to Level of Control. *Robert L. Jackson, Robert C. Hardin, Glenn L. Walker, Atlee B. Hendricks, and Helen G. Kelly, State University of Iowa, Iowa City, Iowa.* Pediatrics 5: 959–71, June 1950.

This is a study of the degenerative changes in 75 juvenile diabetic

patients who have had their disease over 10 years while under close supervision. There were 40 boys and 35 girls. In every one the disease had appeared before 14½ years of age. In approximately one half, the duration of the disease was more than 15 years; in 11, it was over 20 years. The ages at the time of last examination ranged from 13 to 32 years (average, 22.9 years). All had severe diabetes as measured by insulin dosage and insulin weight ratio.

These patients had been under the care of a pediatric clinic in which a good level of control was always maintained. The diets given during early childhood and the prepubescent spurt and for retarded growth contained approximately 1.5 Gm. of animal protein and 35 to 40 calories per pound of theoretic weight. The diets during midchildhood contained approximately 1 Gm. of animal protein and 30 calories per pound of theoretic weight. Amorphous regular insulin and a slowly acting insulin (globin insulin with zinc) had been given. With few exceptions, hospitalization was necessary only at the first observation in order to determine the insulin requirement of the patient. The 24-hour requirement of insulin ranged from 30 to 108 units. The average for the group was 60 units at the time of last examination. Twenty-two per cent of the cases were classified as very severe, 60% as moderately severe, and 18% as less severe.

The levels of control of the disease were rated as follows:

Control rating 0 (Good)—Freedom from sugar in the urine except for occasional slight traces. Approximately normal blood sugar levels.

Control rating ½ (Fair to good)—Fluctuating from rating 0 to 1.

Control rating 1 (Fair)—Less than one half of urine specimens free from sugar and small amounts of sugar in the remaining specimens. Slightly elevated blood sugar levels.

Control rating 2 (Fair to poor)—Fluctuating from rating 1 to 3.

Control rating 3 (Poor)—Varying amounts of sugar in urine continuously. Elevated blood sugar levels.

Control rating 4 (Very poor)—Continuous gross glycosuria. Elevated blood sugar levels.

The most recent physical examinations disclosed that, with few exceptions, these patients were enjoying good health. Four exhibited diseases not directly related to diabetes mellitus. More directly related were minimal obesity in 7 patients and retarded physical development in

TABLE 1—*Type and Incidence of Degenerative Changes in 75 Juvenile Diabetic Patients*

	Males	Females	Total	Per Cent
Retinopathy	22	13	35	46.6
Cataracts	6	5	11	14.6
Calcification of blood vessels	6	6	12	16.0
Hypertension	2	1	3	4.0
Nephropathy (albuminuria)	1	2	3	4.0
Abnormal ECGs	0	0	0	0

2; 2 others had hepatomegaly with evidences of hepatocellular dysfunction. Table I lists the degenerative changes found in the last examination of these 75 juvenile diabetics.

The comparisons showed that in poorer control and in duration this disease was correlated with retinopathy, calcification in the peripheral arteries, hypertension, proteinuria, retardation of growth, and recurrences of acidosis and ketosis. Higher values for serum cholesterol and total lipids were more common in patients with poorer levels of control. Only those who maintained excellent levels of control had values comparable to nondiabetic subjects. More than one degenerative abnormality frequently occurred in a single patient. There was no correlation between the incidence of early or advanced cataracts and the level of control or the duration of the disease. Cataracts seemed to appear chiefly in those patients who had suffered short periods of marked noncontrol, including acidosis.

The conclusions were drawn that the level of control and the duration of the diabetes are important factors influencing degenerative changes. Good control will delay and possibly prevent degenerative changes. It is not only possible but practicable to maintain a high level of control of diabetes in children living in a well adjusted home. Until more knowledge is available, a high level of control appears to offer the best means of averting or delaying chronic degenerative changes.

More information is needed to understand the pathologic physiology of various levels of noncontrol. An optimistic rather than a pessimistic outlook for juvenile diabetes is warranted. 25 references. 4 tables.

Lesions of Galactose Diabetes. Pathologic Observations. *Leo S. Bell, William C. Blair, Stuart Lindsay and Standish J. Watson, University of California Medical School, San Francisco, and Mills Memorial Hospital, San Mateo, Calif.* Arch. Path. 49: 393–403, April 1950.

The authors add a description of the pathology of 2 cases of galactose intolerance ("galactose diabetes") in siblings, to the 16 instances already on record in medical literature. One of the patients died at 8 days of age, and the other recovered when lactose was eliminated from the diet.

The essential clinical features of this disease are galactosuria, galactosemia and hepatomegaly, with icterus as a common complication. The enlargement in the liver may lead to portal vein obstruction with ascites, splenomegaly, and engorgement of the abdominal veins. An extreme degree of intolerance to galactose in an infant on a milk diet may lead to an early death, whereas a milder disturbance can eventuate in severe malnutrition, with secondary mental retardation or lamellar cataracts. The pathologic changes are reversible in the early stages. The galactosuria, galactosemia, and liver symptoms can be eliminated by cessation of the feeding of milk which is the source of the galactose. This disease is considered by some as a variant of glycogenosis or von

Gierke's disease, but of reversible character. Only two histologic reports of the liver lesions have been recorded, with the liver appearing cirrhotic or malformed.

A description is given of the postmortem findings in the fatal case and of the liver biopsy findings in the sibling which survived. There were no significant lesions in the organs other than the liver. There was pronounced fatty infiltration of the hepatic parenchymal cells of the infant who died at the age of 8 days, with some foci of necrosis and prominent fatty infiltration. The liver of the other infant, who was slightly older [age at biopsy was not stated], exhibited active parenchymal cellular necrosis with acute inflammation. Numerous parenchymal cells had disappeared, allowing the supporting stromal framework to collapse and become condensed. No active fibroblastic proliferation was observed at this period, but this pathologic state was regarded as early cirrhosis, which might be expected to progress if the fatty infiltration persisted. Both livers contained abundant lipid material, and no excess of glycogen.

From these findings it is concluded that the hepatomegaly that occurs with galactose diabetes is the result of fatty infiltration of the liver, and that the secondary degenerative changes in the hepatic cells may lead to hepatic cirrhosis. This liver lesion appears to be reversible if treated early by elimination of dietary galactose, irreversible if excessive degeneration and scarring take place. 11 references. 4 figures.

See also next abstract—EDITOR.

Galactose Diabetes (Galactosemia). A Clinicopathologic Study of Two Siblings. *Leo S. Bell, William C. Blair, Stuart Lindsay and Standish J. Watson, Mills Memorial Hospital, San Mateo, Calif., and University of California Medical School, San Francisco, Calif.* J. Pediat. 36: 427–39, April 1950.

This is the report of the clinical histories of 2 siblings with congenital galactose intolerances. One died at the age of 8 days with bleedings, coma, hepatomegaly, and jaundice, and the diagnosis was made in retrospect.

The second sibling had jaundice, vomiting, drowsiness, hepatomegaly and splenomegaly commencing in the first week of life. Laboratory studies were negative except for serum icterus and glycosuria in the absence of ketonuria. Studies of the urine sugar revealed it to be galactose. The fasting blood sugar was 218 mg. per 100 ml. When repeated it was 173 mg. per 100 ml. of which 144 mg. were nonfermentable reducing substances, presumably chiefly galactose. The galactose tolerance test was abnormally prolonged. An injection of 0.2 ml. of epinephrine produced a rise in blood glucose and total blood sugar and a slight fall in blood galactose. Following each intravenous injection of galactose the infant appeared to be ill, apathetic, and was anorexic. There was fever after each injection which persisted for 24 hours. An intravenous glu-

cose tolerance test revealed excellent utilization of glucose. Liver function tests revealed minor changes.

Because there was intolerance to ingested galactose the child's diet was changed from milk to a protein-hydrolysate-containing milk substitute. Within a few days the urinary sugar and albumin disappeared, and the child started to gain weight. The symptoms disappeared, and growth thereafter was normal. Some weeks later a milk diet given for 24 hours produced a transient galactosuria once more.

It seems apparent from these experiences and the cases reported to date that early diagnosis and prompt institution of appropriate treatment offer these children a good prognosis, since the early manifestations of the disease are reversible. If treatment is instituted late, changes will develop. These include cataracts, severe malnutrition, hepatic damage or cirrhosis, and mental deficiency. 22 references. 2 figures. 3 tables.

This disease is obviously a congenital error of metabolism. One questions whether the difficulty is limited strictly to the single carbohydrate galactose.

Attention may be directed to the recent isolation of the coenzyme of the galactose phosphate-glucose phosphate transformation by Caputto, Leloir, Cardini and Palladini (J. Biol. Chem. 184: 333–50, May 1950). The principal components of this newly isolated substance are uridine, two phosphate groups, and glucose. It has therefore been named uridine-diphosphate-glucose (UDPG). The chain of chemical reactions by which galactose can be converted into glucose within body tissues is comparatively short, but requires the presence of several enzymes and coenzymes—galactokinase, glucose diphosphate, a "galactowaldenase," and the newly isolated UDPG. It is highly plausible that UDPG or one of the other essential enzymes or coenzymes is missing in congenital galactosemia.—Editor.

17. Milk; Infant and Child Feeding

The Role of Human Milk in the Transmission of Antibodies. *Bo Vahlquist, Stockholm, Sweden.* Acta. pædiat. Suppl. 77: 56–60, 1949.

Antibodies may be transmitted to the offspring in two ways: either in utero via the blood or after parturition via the milk. The predominant route is different in different species. In man the placenta is permeable to antibodies. For many types of antibodies the concentration is the same in the blood of the newborn as in the blood of the mother. The antibodies in the colostrum and the milk reflect those in the mother's blood, but the concentrations are, as a rule, much lower.

To what extent the infant many absorb the antibodies in milk is still a matter of discussion. In collaboration with Högstedt the author performed experiments dealing with the absorption of diphtheria antibodies. High titer homologous serum was fed for several days in

amounts varying between 25 and 150 A. U. per Kg. of body weight. The antitoxin was administered from the first to the fifth day of life to 13 newborn infants. Absorption could be demonstrated in 9 of these, but the amount absorbed never exceeded 0.3% of the quantity given. In 14 infants above the newborn period absorption could be traced only in 2 cases, and only in minute amounts. 3 figures.

High Protein Nutrition in Prematures. *Arne Rothe-Meyer, Copenhagen, Denmark.* Acta. pædiat. Suppl. 77: 109–11, 1949.

Considerable evidence is available to indicate that premature infants thrive better on milk formulas containing more protein and less fat than on breast milk alone. Original studies are referred to in which premature infants given an undiluted half-skimmed citric acid milk had a greater rate of weight increase than another group fed on human milk.

The blood urea levels in the prematures fed solely on human milk during the first two weeks of life varied between 5 and 40 mg. per 100 ml. This variability is probably due to the functional immaturity and tendency to dehydration during the neonatal period. At the age of 4 weeks the majority of urea values fell between 5 to 15 mg. per 100 ml. The urea levels in the prematures fed on half-skimmed citric acid milk had a similar large variation at a somewhat higher level (10 to 40 mg. per 100 ml.) in the first two weeks. But unlike those on human milk, those on cow's milk continued at this higher level (20 to 40 mg.) until and after the age of 4 weeks, and not infrequently reached still higher values. It was concluded that the higher blood urea in levels of premature infants fed on half-skimmed citric acid milk represent increased functional strain. 1 table.

18. Miscellaneous

The Third Fontanel (*La troisième fontanelle*). *M. Forest, Strasbourg, France.* Nourrisson 38: 95, March–April 1950.

Some newborn infants, in addition to the usual fontanels, have a separation of the edges of the parietal bones, which the author designates as the third fontanel. This used to be rarely observed. After July 1945, in the postwar period when infantile mortality showed a marked increase, the author observed this separation frequently, probably as a sign of the general physiologic deficiency of the newborn at that time. In the past two years it has again become much less common.

19. Musculoskeletal System

Rheumatoid Arthritis in Young Children Treated with Gold. *R. K. W. Kuipers and M. F. Schouten, 's-Grevenhage, The Netherlands.* Nederl. tijdschr. v. geneesk. 93: 4235–42, Dec. 17, 1949.

Nineteen children suffering from rheumatoid arthritis were treated with gold (solganal-boleosum). The initial dosage was 5 mg. which was

slowly raised to 50 or 80 mg. according to age and body weight. Physical therapy was an important aid. Treatment was started at the average age of 7 years. Some cases could be followed seven years after the first course was completed. Some relapses were noted. A new course with gold salts brought a favorable result again. The authors view their results as "good" in 12, "moderate" in 4, and "bad" in 3. Two of these died. The clinical history of 3 patients is reported in detail. 5 references. 1 figure.—*F. Kuipers.*

20. Nervous System

The Neurological Sequelae of Rh Sensitization. *P. R. Evans and P. E. Polani, Guy's Hospital, London, England.* Quart. J. Med. 19: 129–50, April 1950.

The outstanding neurologic sequels of erythroblastosis fetalis caused by Rh sensitization are described as seen in 16 original cases and 63 others selected from literature. In the neonatal period the prominent signs are illness, jaundice, opisthotonus and muscular twitchings. Convulsions, rigidity, muscular spasms, a cerebral cry, or a bulging fontanelle are less regularly seen. A latent period of several weeks or months then ensues, to be followed by stiffness or hypotonia. Mental defect is often but not always present. Deafness is frequent.

Muscular spasticity is noted at all ages; this varies from classical spasticity with stretch-reflexes, through plasticity, to muscular spasm in extension which is especially noticeable on manipulation. Typical features of spastic paraplegia or spastic diplegia are very uncommon. Muscular flaccidity does not become noticeable until the end of the first year. It is more obvious in the trunk and neck than in the limbs, and disappears gradually during the ensuing years. Irregular movements may be noted early. After the third year, most patients have involuntary movements, usually athetoid or choreic, and associated with spasm. Muscular spasm usually appears before or with the athetosis. The choreic movements seem to be much more common in infantile cerebral palsy resulting from Rh incompatibility than from all other causes combined. These movements, which are rapid and jerky, may not begin until the child is several years of age. Convulsive movements may occur in the neonatal period and through the first year, but are rare later. Epilepsy and blindness are rare sequels, but deafness is common; this may be partial or complete.

From a correlation of the autopsy reports and clinical findings it is apparent that most of the central nervous system damage in this disorder takes place in the cerebral cortex, the subthalamic body and the globus pallidus. 111 references. 2 figures. 1 table.

The growing number of neuropathologic studies on erythroblastosis fetalis are making it increasingly clear that the injury suffered by the brain is a nonspecific change, similar if not wholly identical with that produced by various other conditions which cause anoxia. The nerve

cells are injured reversibly or irreversibly, with the ganglion cells in the cortex and some of the higher centers showing the most severe changes. Capillary hemorrhages and secondary scarring are frequently seen. Intravascular agglutination with thrombosis is extremely uncommon if it occurs at all.

The yellow staining of selective regions, known as kernicterus, is somehow closely related to the nerve damage, but the precise relationship is not clear. Neither the cortical damage nor the yellow staining are seen in stillborn erythroblastic infants. There is a growing tendency for clinicians to use the term "kernicterus" when referring to the central nervous system deterioration exhibited by survivors of erythroblastosis, but proof has not yet been adduced that all such injured nerve tissue passes through a phase of yellow staining on being injured.—EDITOR.

Pseudotetanus, a Forgotten Syndrome (*Pseudo-tetanus, een vergeten ziektebeeld*). *E. den Hartog-Ricardo, Haarlem, The Netherlands.* Maandschr. v. Kindergeneesk. 17: 59–62, 1949.

In 1896 Escherich described a form of tetany occurring in boys which he called pseudotetanus. It was characterized by tonic contractions of the lower extremities, trunk and face, with trismus and risus sardonicus. The musculature of the upper extremities was hardly involved or completely free. The disease healed in a few weeks. Subsequent authors felt that it might be a form of encephalitis.

The case of a 10-year-old boy is described, with trismus, risus sardonicus and marked hypertonicity of all muscles except those of the arms. Reflexes of the legs were hyperactive, those of the arms normal. The electric irritability of the affected muscles was decreased. The spinal fluid was normal as well as the blood calcium. There was no indication of tetanus; there were no true muscular contractures. The disturbance subsided without treatment; after four weeks nothing abnormal could be found. The author was of the opinion that the diagnosis of pseudotetanus was appropriate.—*Ph. Arons.*

21. Newborn Period, Prematurity

Physiologic Background of Fetal Anoxia at Birth and Cyanosis in the Newborn. *Clement A. Smith, Boston, Mass.* Am. J. Dis. Child. 79: 1–9, Jan. 1950.

The relationships between fetal and neonatal anoxia and cyanosis in the newborn infant are discussed with regard to physiologic background and the observable results of oxygen administration. Anoxic anoxia, characterized by low oxygen tensions in arterial as well as venous blood, may occur in fetal life or at birth because of (1) maternal pulmonary or circulatory disease; (2) anesthetic gas replacing maternal oxygen; (3) interference with placental exchange or umbilical cord circulation, and (4) impaired aeration of blood in the neonatal lung from numerous

central nervous system and pulmonary causes disturbing respiratory efficiency. Cyanosis, which frequently accompanies anoxic anoxia from any of the above causes, should be to some extent ameliorated by increasing the oxygen of the atmosphere breathed. On the other hand, when anoxic anoxia occurs: (5) from shunts from right to left due to congenital cardiac or vascular anomalies, oxygen inhalation is unlikely to affect the cyanosis usually present. Stagnant anoxia, characterized by too great reduction of oxygen tension in the capillaries, is commonly present in the extremities of newborn infants because of: (6) sluggish circulation from cold or other causes. The cyanosis observed either locally or generally will not improve by oxygen administration unless the stagnation is itself a result of circulatory failure from one of the forms of anoxic anoxia listed above as so improvable. Anemic anoxia may occasionally be produced by: (7) neonatal hemorrhage; (8) replacement of functional hemoglobin by methemoglobin as a consequence of exposure to aniline dyes, nitrates, or bismuth. In (7) cyanosis is unlikely to occur; in (8) it occurs because of the methemoglobin, but this will be little affected by oxygen administration.

Breathing at birth may well begin in the unimpaired infant as a response to peripheral sensory stimuli. If the infant is physiologically impaired (by trauma, drugs, or anoxia), respiration of a more primitive nature may be induced by the mounting anoxia (or hypercapnia, or both), and, having so originated, may improve in response to oxygen administration. This hypothesis, drawn largely from Barcroft's work on animals, seems to apply well to observations of newborn infants. 8 references. 3 figures.—*Author's abstract.*

Death of Cesarean Infants: A Theory as to Its Cause and a Method of Prevention. *Daniel B. Landau, Howard B. Goodrich, W. F. Francka and Francis R. Burns, Hannibal, Mo.* J. Pediat. 36: 421–26, April 1950.

The mortality among cesarean-delivered infants is generally reported to be near 9%. The authors have been impressed by such infants who leave the operating room in apparently good condition but later, in whom cyanosis, respiratory distress with dyspnea, air hunger, costal retraction, and rapid weak pulse develop. They sink rapidly and die in terminal convulsions in about 18 to 20 hours, despite all supportive measures.

It is suggested that the crucial point of difference between the normally delivered and the cesarean-delivered infant lies in the handling of the umbilical cord and placenta. The immediate clamping and cutting of the umbilical cord deprives the cesarean-delivered infant of blood which he would ordinarily receive under conditions of normal delivery, so that the shock symptoms here described may occur. Ballentine had found that delayed clamping of the cord increased the amount of blood the newborn infant received by 96 cc. on the average. DeMarsh, Windle

and Alt found that more than 26% but less than 34% of the average fetal blood is in the placenta and umbilical vessels at the end of fetal life. Under normal conditions of delivery most of this blood passes into the infant during the third stage of labor while the uterus is contracting on the placenta.

Because of these considerations the authors altered their technic of cesarean sections to provide more blood for the infant. At the time of delivery the assistant designated to care for the infant is scrubbed and stands to the left of the operator. When the infant is removed from the uterus this assistant holds the infant by the legs with the head down while the cord remains attached to the placenta. The pharynx is aspirated by means of a rubber bulb syringe. The placenta is separated from the uterus and wrapped in a large turkish towel. Several clamps are used to secure the placenta because it slips easily. The infant and wrapped placenta, still connected by the intact umbilical cord, are placed on another table. The placenta is suspended from a standard by means of a clamp attached to the towel, while the infant rests on the table, wrapped in sterile cloths and warm blankets. Stimulants of choice and an infant resuscitator are in readiness. After its blood vessels have collapsed, the cord is clamped, cut, and tied in the usual manner. It takes 6 to 10 minutes for the placenta to drain and the cord to collapse.

In 87 cesarean sections done since this technic was instituted, the secondary shock phase has not been observed. Two deaths occurred, neither due to a shock-like syndrome. The series included two premature infants, one with placenta previa and the other with uteroplacental apoplexy. Both of these survived.

This procedure seems especially valuable with premature infants since the placenta in relation to body size is larger in the premature than in the mature baby and the quantity of placental blood is greater in relation to the infant's blood volume. 9 references. 1 table.

A promising suggestion.—Editor.

Failure of Vitamin B_{12} to Promote Growth of Premature Infants. *D. F. Downing, Hahnemann Medical College, Philadelphia, Pa.* Science 112: 181, Aug. 1950.

Twenty-five of 48 prematurely born infants were given vitamin B_{12}, the remainder serving as controls. The weight of the entire group was between 1245 to 2326 Gm.; the average weight of the experimental and control groups was approximately the same. The dose of vitamin B_{12} was 10 μg. intramuscularly every 1 to 3 days beginning on the second to the seventh day of life. The total amounts given ranged from 30 to 220 μg. (average 70 μg.). There was no difference in the daily or total weight gain between the two groups or in the average time to reach 2.5 Kg. In fact, a slight superiority was noted in favor of the controls. It was concluded that B_{12} will not benefit the growth of premature infants unless there is a specific deficiency. 1 table.—*A. M. Bongiovanni.*

Tonic-Neck-Reflex and Symmetro-Tonic Behavior. Developmental and Clinical Aspects. *Arnold Gesell, and Louise B. Ames, New Haven, Conn.* J. Pediat. 36: 165–76, Feb. 1950.

Magnus in 1926 described the tonic neck reflex (t-n-r) as observed in experimental animals (rabbit, cat) and in neuropathological human subjects. Gesell in 1934, 1938 and 1945 reported evidence that the t-n-r pattern is a normal and prominent form of spontaneous postural behavior in fetal infants, and in fullterm infants, throughout the first twelve weeks of life. Most often the head averts to the right. The right arm is typically held (or activated) in lateral extension, the opposite arm being flexed at the left shoulder, simulating a fencing attitude. By 16 weeks the head is more mobile. At 20 weeks 100% of a group of normative (supine) infants maintained the head in the mid-position, symmetro-tonic attitudes (s-t-r) superseding the asymmetric: the arms adduct and flex simultaneously toward the midplane to contact an object or to engage in mutual fingering. Simultaneous bilateral approximation of the arms has also been observed in the fetus.

The t-n-r is not a stereotyped, unalterable entity. It is subject to growth changes in the normal child. Marked persistence of the t-n-r beyond the first half year is indicative of retarded, arrested, or defective development. Frame by frame analysis of motion picture records of extreme athetosis in a 7-year-old boy revealed the presence of the t-n-r, in association with a basal ganglion lesion. In normal development the quiescent and the activated t-n-r promotes and channelizes visual fixation by the infant on his extended hand; it leads to hand inspection, to eye-hand coordinations, to prehensory approach, and eventually to unidextrality.

Unilateral, bilateral, ipsilateral and contralateral members must be brought into parallel and diagonal coordinations, and to conjunctions and dysjunctions in functional timing. This involves an enormous amount of ontogenetic dove-tailing, a pervasive process of neuromotor interweaving. The principle of reciprocal interweaving which underlies this process is formulated as follows: The organization of reciprocal relationships between two counterpoised sets of interacting functions is ontogenetically manifested by more or less periodic shifting ascendancies of the component functions with progressive integration and modulation of the resultant behavior patterns.

Viewed in terms of the principle of reciprocal interweaving, the asymmetric and symmetric postural behavior patterns of the human infant assume far-reaching clinical significance. Normality of neuro-motor development depends upon lawful timing and balance of the asymmetric and symmetric manifestations. Faulty ontogenetic timing, cerebral injury, and constitutional deviations genetic in origin likewise account for mixed and poorly defined dominances which come to ultimate expression in visual inadequacies, reading and speech disabilities, and

neurological symptoms. For all these reasons tonic-neck-reflex and correlated symmetro-tonic phenomena are entitled to continuing investigation and to routine clinical scrutiny. 17 references. 2 figures. 2 tables. 2 graphs.—*Author's abstract.*

22. Nutrition

Nutritional Requirements During Infancy. *Genevieve Stearns, State University of Iowa, Iowa City, Ia.* J. Iowa State M. Soc. 40: 154–59, April 1950.

The first nutritional requirement of the infant is that the mother be well nourished during gestation and preferably also before pregnancy. Infants born of well-nourished mothers are superior physically to those whose mothers were poorly fed, and remain so during most of infancy.

Requirements for specific nutrients in infancy are conditioned by the fact that the energy, protein and mineral requirements are high, on a weight basis, because of the rapid growth rate. The young infant's kidneys do not concentrate well; the younger and more immature the infant, the greater is his relative water requirement. The very young infant has a limited ability to oxidize fat, so for premature, immature and young infants, the amount of fat fed is limited and extra calories are provided from carbohydrate.

The requirement of the fullterm infant for calories, protein, calcium and phosphorus, sodium, potassium, water, riboflavin, and vitamin A will be met when the breast-fed infant receives 2 to 2½ ounces of milk per pound and the infant fed cow's milk gets 1½ to 2 ounces per pound of body weight of milk containing 6 to 8% added carbohydrate. The requirements for thiamine and niacin are probably met. Vitamins C and D must be provided, preferably as soon as feeding is instituted: additions of foods containing iron and thiamine are desirable by 3 months or soon thereafter.

As nearly half of the protein and calcium of the newborn infant is deposited during the last 4 weeks of gestation, the prematurely born infant is greatly handicapped. With his limited gastric capacity, it is impossible for him to get sufficient of these nutrients from human milk alone. It is therefore customary to add dry skim milk or calcium caseinate to the human milk-fed premature infants. Their need for vitamin D is no greater than that of the fullterm infant.

Premature infants normally have a high and very labile water content, therefore they are peculiarly liable to both edema and dehydration. They cannot concentrate urine, so the water requirement is high. Their enzyme systems are incomplete and they excrete relatively large amounts of organic acids, which they cannot oxidize. They are thus always mildly acidotic. Vitamin C in larger amounts than is needed for fullterm infants aids in the oxidation of phenylalanine.

Overdosage with fat-soluble vitamins A and D can and does occur with both fullterm and premature infants and should be avoided. Four hundred units of vitamin D daily provide maximum retention of calcium and phosphorus and more rapid rate of growth than amounts much higher or lower than this level. Intakes of 2,000 units daily are mildly toxic. The toxic level of vitamin A is not known but is much higher than that of vitamin D. Nevertheless, acute toxicity has occurred from overdosage. Amounts of vitamin A up to 3,000 I.U. daily (5 to 6 times the vitamin D dosage), may be added to the feeding with impunity, as far as effects on growth, development, or appetite of fullterm infants are concerned. 28 references.—*Author's abstract.*

Hypervitaminosis A. *Charles T. Fried and Milton J. H. Grand, Bronx Hospital, New York, N. Y.* Am. J. Dis. Child. 79: 475–86, March 1950.

Two children are described whose clinical histories conform with the criteria deemed necessary for establishing the diagnosis of hypervitaminosis A. These criteria are: (1) a history of excessive intake of vitamin A over a prolonged period, (2) a symptom complex which is consistent with reported findings in animals and other human cases, (3) a steady and complete recovery after withdrawal of vitamin A, (4) roentgenographic bone changes similar to those resulting from excessive vitamin A in animals, and (5) elevated vitamin A levels in the blood serum. One patient was a 16-month-old boy who had received 2 teaspoons (8 to 10 ml.) of percomorph liver oil daily for over a year. The other, a 2-year-old boy, had been given ½ to ¾ teaspoon (2 to 3 cc.) of percomorph liver oil daily for many months, in addition to huge amounts of butter. This child would often eat ¼ to ½ pound (113 to 226 Gm.) of butter at one session.

Cases of this sort ought to be more prevalent in view of the widespread use of concentrates containing vitamin A and the early introduction of foods high in vitamin A and carotene content. It is therefore suggested that other factors than simply the ingestion of large amounts of vitamin A may be involved in the pathogenesis of hypervitaminosis. For the development of this syndrome there may have to be a faulty metabolism of the ingested vitamin, a dysfunction of the liver, or a disturbance in the storage, destruction or excretion of vitamin A.

The injurious effects which follow the ingestion of excessive amounts of vitamin A in human beings include anorexia, loss of weight, irritability and fretfulness, hepatomegaly, low-grade fever, pruriginous rash, sparseness of hair, and extreme tenderness of the long bones to pressure. There are abnormally high vitamin A levels in the blood serum and the serum lipids are increased. The long bones exhibit localized periosteal swellings in the long bones and pain in these areas; there may be irregular epiphyses, lateral bowing of the bones, and widening of their ends.

The vitamin A level in the blood determined in the first patient was 1,630 I.U. while he was receiving the percomorph oil. One month after the oil was discontinued the fasting level was 294 I.U. The absorption curve after ingestion of 65,000 units of vitamin A gave readings of 465, 444 and 411 units, at 3, 6 and 9 hours, which is a normal curve except for a high fasting level for vitamin A (normal range, 30 to 100 units). The curve in the second patient, determined two weeks after vitamin A was discontinued, showed a fasting level of 1,600 units, with readings of approximately 2,000, 3,750 and 2,500 units at 3, 6 and 9 hours. Vitamin A is ordinarily not excreted by the kidney in the human being unless the amount ingested is huge, and then only under abnormal conditions. The urine in the second patient contained 1.3 units per 100 ml.; the stool 24 units per Gm. which is normal.

Most healthy individuals tend to have a more or less constant level of vitamin A in the blood regardless of fluctuations in intake. The constancy of this level seems to be maintained by the liver, which can carry a relatively enormous reserve. The content in the liver is directly related to the intensity and duration of the intake. No harm to the body results from this storage, it is believed, until the capacity to keep the blood level stable becomes overwhelmed. The blood concentration will not fall to an unusually low level until after the liver stores have become depleted. A sudden large dose of vitamin A raises the plasma level beyond the normal maximum for only a few hours. An excessive intake over a long period of time can lead to persistently high levels of abnormal degree and the appearance of toxic symptoms, as the result, apparently, of failure of the regulatory mechanisms. Some children seem to be able to resist the development of hypervitaminosis A. The 8-month-old infant cited by Josephs had been given 500,000 units of vitamin A daily for 4 months yet after this time disclosed no clinical evidence of toxicity and had a normal vitamin A blood level of 110 units. The only available test for estimating the ability of the liver to metabolize excessive doses of vitamin A is the vitamin A tolerance curve. 22 references. 4 figures.

23. Parasitic Diseases

Monilial Granuloma: Report of a Case and Review of the Literature. *Fred V. Hauser and Stephen Rothman, University of Chicago, Chicago, Ill.* Arch. Dermat. & Syph. 61: 297–310, Feb. 1950.

Monilial granuloma is a rare specific disease entity resulting from Candida albicans infection. It begins in infancy or early childhood, often as simple oral thrush, and ultimately involves three sites: the oral mucosa, the finger nails and paronychial tissues, and the skin of the scalp and face. The skin of the trunk and extremities is rarely involved. The sexes are involved equally, and it occurs in whites and Negroes. In contrast to the banal superficial forms of moniliasis, monilial granuloma

is characterized by an inflammatory reaction extending into the corium, and consisting of granulation tissue similar to that in the deep mycoses. These conclusions are based on observation of 1 case by the authors which is described in detail and on reports of 13 cases uncovered in the literature.

The primary cutaneous lesion is a highly vascularized papule about 5 mm. in diameter covered with a thick, adherent yellow-brown crust. Plaques covering large portions of the face and scalp are formed by confluence of the papules. Horns formed by accumulation of the scales and crusts surmounted the papules in about half the cases. These are regular columnar structures on the glabrous skin; on the scalp they are intermeshed with hairs and curved to follow the fall of the hairs. The bases of the cutaneous lesions consisted of friable granulation tissue which extended in digitations along the hair shafts. The clinical picture often suggested favus.

The lips and buccal mucosa were involved in nearly all cases. The lesions of the lips varied from erosions and rhagades at the mouth corners to severe painful swelling and crusting, with deep fissures between the crusted areas. The buccal mucosa and palate are involved to variable degrees with erosions and pseudomembranes, as in thrush, or show numerous hyperkeratotic areas which bleed profusely if scraped.

In all cases the distal phalanx of one or both thumbs was swollen and the nails greatly heaped up into rough, opaque pyramidal structures. Other fingernails were also involved. The toe nails were not affected. Here, the infection begins proximally with painless, nonpurulent swelling of the paronychial tissues; subsequently the lunula is involved and ultimately the entire nail plate becomes permanently altered. The regional lymph nodes are often swollen and tender. The hair or lesions do not fluoresce under filtered ultraviolet light.

The causative fungus, Candida albicans, grows rampantly in all the infected sites, but not inside the hair. Direct examination of scrapings reveals innumerable fine, straight, poorly refractile hyphae, and clusters of spores. Cultures of material taken from all involved sites and sputum and stools yielded typical colonies of Candida albicans. Histologic examination revealed hyperkeratosis and acanthosis, and a dense chronic granulomatous reaction in the corium. The disease has never been transmitted to other persons in this form.

The prognosis is poor. It is usually resistant to all forms of therapy, although the skin lesions do show partial healing with fine pitted scars. Four patients who were observed for a considerable period of time died before the age of 20, 1 of miliary tuberculosis, 2 during an attack of a chronic recurrent pulmonary infection, and 1 of typhoid fever. However, the latter patient was found at autopsy to have "multiple masses diffusely scattered about both lungs." Unfortunately in the latter 3 cases it was never determined whether the patient suffered from generalized moniliasis. It is concluded that this unusual manifestation of

an infection by Candida albicans is due to increased susceptibility of the host, rather than increased virulence of the infecting organism. 11 references. 6 figures.—*Author's abstract.*

The Efficiency of the Treatment of Active Oxyuriasis with Vermifuges In General Practice (*De doelmatigheid van de behandeling van actieve oxyuriasis met vermifuga in de practijk*). *J. C. de Wit, Scheveningen, The Netherlands.* Nederl. tijdschr. v. geneesk. 93: 1234–40, April 16, 1949.

The action of gentian violet and phenothiazine on oxyuriasis was studied. Adults received 65 mg. of gentian violet three times daily for two periods of 8 days each, with an interval of 1 week; children received 15 mg. per year of age over the same periods. The dosage of phenothiazine was 50 mg. per Kg. body weight per day at all ages, given in three or four doses each day for 2 days. Both drugs were given in two courses, with an interval of 3 weeks. If the stools contained ova 5 weeks after the last day of treatment, this was considered to be a relapse. If the stools became positive after 14 weeks, a reinfection was assumed. In the interval between the fifth and the fourteenth week the finding of ova may signify either a relapse or a reinfection. Treatment with gentian violet was followed by relapse after 5 weeks in 84 to 90% of cases, whereas phenothiazine treatment was followed by relapse in only 5 to 8%. The percentage of reinfection and/or relapses was 88 to 95% in the gentian violet groups, and 8 to 20% in the phenothiazine groups. After a year the percentages were 96 to 100% and 88 to 90%, respectively. Phenothiazine should be reserved for the serious cases of oxyuriasis as it may be toxic even in the dosage used. The toxic effects are: vomiting, transient jaundice, slight hemolytic anemia, albuminuria. The drug should not be given to any patient suffering from a disease of the liver, spleen, kidneys or hemopoietic organs. Because reinfection is so common, oxyuriasis is more a hygienic than a pharmacologic problem. 21 references. 1 table.—*M. G. Stronk.*

24. Pathology, Anatomy, Bacteriology

See Contents for Related Articles

25. Physiology, Biochemistry

Idiopathic Hypoparathyroidism with Papilledema in a Boy Six Years of Age. Report of a Case Associated with Moniliasis and the Celiac Syndrome and a Brief Review of the Literature. *C. Collins-Williams, Harvard Medical School, and the Children's Medical Center, Boston, Mass.* Pediatrics 5: 998–1007, June 1950.

At 16 months of age a normally developed boy was seen with moni-

liasis of the mouth caused by Candida albicans. Treatment seemed to clear the oral lesions, but the infection recurred in the mouth and nails over the next few years. At 5 years of age he began to pass loose, foul-smelling stools and the diagnosis was the celiac syndrome. At 6½ years he was hospitalized in severe tetany and found to have hypoparathyroidism. His blood calcium level was 5 mg. per 100 ml., the phosphorus 6.4 mg. and the alkaline phosphatase 6 Bodansky units. A modified Ellsworth-Howard test demonstrated an increased output of urinary phosphorus after parathyroid extract, indicating that the patient could respond to parathyroid hormone and therefore did not have pseudohypoparathyroidism. The bones were normal by roentgenogram and no intracranial calcification was observed. While he was in the hospital, bilateral papilledema developed, then receded, apparently in response to therapy for the hypoparathyroidism. Treatment consisted of calcium gluconate intravenously and large doses of calcium gluconate, water-soluble vitamin D, AT-10 and creamalin by mouth, as well as a high protein, low fat, low phosphorus diet. The stools became normal shortly after this diet was started.

The 22 previously reported cases of papilledema associated with hypoparathyroidism and the 7 previously reported cases of moniliasis associated with hypoparathyroidism are briefly reviewed. One brother, in whom moniliasis developed at 1½ years of age and the celiac syndrome at about 3 years, still has at 9 years no evidence of hypoparathyroidism. Four other siblings are well. 18 references. 1 figure. 2 tables. 3 charts.

Tests for evaluation of the functional state of the parathyroid glands in a patient are few and as a rule controversial. One of the most useful in evaluating hypoparathyroid states is the phosphorus diuresis test, perhaps better known as the Ellsworth-Howard test. Adults are given 200 units U. S. P. of parathyroid extract intravenously while fasting, and the phosphorus content of the urine is determined hourly for 3 hours before the medication and 3 to 5 hours afterwards. A normal adult or one with hypoparathyroidism will show an immediate rise from the usual fasting output of 10 to 20 mg. of phosphorus per hour to somewhere between 60 and 120 mg. per hour immediately after the test dose of parathyroid hormone. The hypocalcemic patient with "pseudoparathyroidism" is refractory to parathyroid hormone and undergoes no rise in phosphorus output under this stimulation.

Collins-Williams modified this test because of the difficulty of obtaining hourly specimens of urine from a young child. An intramuscular preparation of parathyroid extract was given while the child was on a low phosphorus diet. Three consecutive 24-hour collections of urine were taken, the first being the control. At the beginning of the second 24-hour period a course of injections of parathyroid extract was begun, 50 units every 12 hours. The total inorganic phosphorus in the urine was 250 mg. in the first 24 hours, 548 mg. in the second 24 hours, and 397

mg. in the third 24 hours. A rise of this degree can be interpreted as normal. The potency of the parathyroid hormone preparation used should always be checked by performing the test on a normal curve of response.

Another modification of this test for use with children is to prolong and pool the observation periods, as was done by Lowe and associates. These authors employed a single intravenous dose of 100 units of parathyroid extract and two 4-hour urine collection periods, one before and one after the injection. In four separate tests their patient with refractory "pseudohypoparathyroidism" had 4-hour totals of urinary phosphorus ranging from 23.5 to 109 mg. during the preliminary control periods and from 22.4 to 65 after hormone was given. (See next abstract.)

The growing recognition of the syndrome of pseudohypoparathyroidism has made this phosphorus diuresis test of great value in the differential diagnosis of possible parathyroid disorders.—EDITOR.

Pseudohypoparathyroidism (The Seabright Bantam Syndrome). *Charles Upton Lowe, Albert J. Ellinger, William S. Wright and Herbert M. Stauffer, University of Minnesota, Minneapolis, Minn.* J. Pediat. 36: 1–10, Jan. 1950.

A white girl was first seen at 2 years of age with the physical and chemical abnormalities described as characteristic of pseudohypoparathyroidism. There was a symmetrically obese face, short stubby fingers and toes, bone distortion, fullness of the dorsum of the hands and feet, and moderate mental retardation. The principal chemical abnormalities were persistence of an elevated serum phosphate (7.5 mg. per 100 ml.) with a decreased serum calcium (5 mg.). The serum phosphatase was 5.1 Bodansky units per 100 ml. at 2 years of age, and 35 units a year later. The principal abnormal function test was a failure to produce a phosphate diuresis in response to the intravenous injection of 100 units of parathyroid extract. Therapy with parenterally-administered vitamin D early in life relieved tetany and returned the concentration of serum calcium and phosphate towards normal. At 4 years of age, a diagnosis of pseudohypoparathyroidism was made and therapy at that time with large doses of vitamin D by mouth resulted in a return to chemical normality.

The patient's history was complicated. Significant features included: (1) the probable existence of lifelong steatorrhea, (2) laryngeal stridor at 3 months of age, (3) hypocalcemia and hyperphosphatemia first noted at 4 months of age, (4) return of serum calcium and phosphorus to normal following parenteral administration of large doses of vitamin D, (5) poor calcium intake in the period immediately preceding the appearance of tetany at 4 years, 7 months of age, (6) elevated serum alkaline phosphatase, during a period when there was neither clinical nor laboratory evidence of liver disease, (7) lack of evidence of

osteomalacia in roentgenograms of the bones, but interference with normal trabecular structure, characterized by intermingling of abnormally dense with abnormally rarified areas, (8) presence of unusually dense metaphyseal bone, and finally, (9) failure of appearance of phosphate diuresis following intravenous administration of parathyroid extract.

The authors suggested that the chronic steatorrhea might have caused defective absorption of vitamin D and calcium, which in time led to hypocalcemia and tetany. The poor absorption of vitamin D, the concomitant malabsorption of calcium from a diet low in that element, and the secondary hypocalcemia resulted in prolonged parathyroid stimulation. The refractory state of the renal tubules as respects parathormone might have had as its origin, at least in part, over-stimulation of the tubules by the patient's own parathyroid gland. Several experimental reports are cited by the authors to indicate that tubular refractoriness may result from chronic low calcium intake or prolonged stimulation with parathormone. 15 references. 2 figures. 1 table.

Pseudohyperparathyroid Tetany. *H. Bakwin, W. F. Gorman and S. R. Ziegra, Bellevue Hospital, New York, N. Y.* J. Pediat. 36: 567–76, May 1950.

A 16-year-old girl is described who exhibited the syndrome recently described by Albright, Burnett, Smith and Parsons as "pseudohypoparathyroidism." Such patients exhibit the signs and symptoms of clinical tetany: convulsions, carpopedal spasm, laryngeal spasm, positive Chvostek and Trousseau signs. They have alterations in body configuration suggestive of achondroplasia: short stature, sturdy build, rounded features and irregularities in the lengths of metacarpals and metatarsals. They have soft tissue calcifications, reduced serum calcium and elevated serum phosphorus. Administration of parathyroid extract (200 units) fails to raise the serum calcium or the rate of excretion of phosphorus in the urine, which is the response given by normal and hypoparathyroid individuals (the phosphorus excretion test). Dihydrotachysterol (AT-10) produces improvement in patients with this syndrome, but more slowly than in true hypoparathyroidism. This sluggishness of response to therapeutic agents led Albright to conclude that the metabolic defect is an insensitivity of the receptor end organs. Vitamin D combined with oral calcium seems to be effective as dihydrotachysterol in treatment.

In this patient the serum calcium was 8.1 and 6.7 mg. per 100 ml. on two determinations; the serum phosphorus was 6.9 and 7.5 mg. After daily doses of 10 drops of dihydrotachysterol for two months the symptoms and signs of tetany disappeared. The serum calcium became 10.2 mg. per 100 ml. and the inorganic phosphorus 3.0 mg. The daily dosage was then reduced to 5 drops of dihydrotachysterol. Calciferol, 100,000 units daily, was later given. 13 references. 5 figures. 1 table.

Hypercalcemia Nephropathy and Encephalopathy Following Immobilization. Case Report. *Katherine Dodd, Harry Graubarth and S. Rapoport, Children's Hospital Research Foundation and University of Cincinnati College of Medicine, Cincinnati, Ohio.* Pediatrics 6: 124–30, July 1950.

The immobilization of a previously active individual by fractures or extensive paralysis is often followed by increased protein catabolism and demineralizing activity within the bones. When these responses are prominent one may encounter hypercalcemia, hypercalciuria and related disturbances in the metabolism of calcium and phosphorus. There exists normally in bony tissue an essentially equal balance between the processes of mineral deposition and mineral removal. Severe restriction of movements leads to an unusual release of calcium into the body fluids, from which it must be removed by the kidney chiefly. Hypercalcemia and the possibility of renal injury take place when the kidney cannot excrete the calcium as rapidly as it is being liberated. The urinary excretion of calcium mounts to reach its maximum at about a month after the beginning of the prolonged immobilization, and continues until motion of the limbs is regained. Roentgenograms may not be able to detect the demineralization in its early stages.

A 12-year-old boy is described who was confined to bed with a fractured left leg retained in a cast. The fracture failed to heal properly after several months, with fibrous dysplasia and cyst formation developing at the site of injury. Hypertensive encephalopathy and renal nitrogen retention became noticeable. Large quantities of milk and sodium bicarbonate were given. The serum calcium rose to 14.6 mg. after four months, which led to a fruitless exploration of the neck region for a parathyroid tumor. The child was then transferred to the care of the authors, four months after the fracture.

Examination showed hypertension (180/130), leukocytes and erythrocytes and Escherichia coli in the urine. The serum NPN was 45 mg./100 ml., calcium 14.9, phosphorus 3.7 mg., and alkaline phosphatase 4.9 Bodansky units. Treatment consisted of removal of the cast, a low calcium diet, and intravenous fluids. The serum calcium and phosphorus gradually returned to normal levels and the signs of hypertensive encephalopathy and of renal damage disappeared. The fracture healed. Diffuse brain damage with epileptic convulsions remained as a sequel. 9 references. 1 figure. 1 chart.

The Significance of Chvostek's Symptom in Infants Under Two Years of Age. *Kjell L. Möller and Bertil Söderling, Borås, Sweden.* Acta pædiat., Fasc. 3–4: 318–22, 1949.

Spasmophilia and rickets are now uncommon in Sweden due to intensive preventive work at the Welfare Centers. Nevertheless, Chvostek's sign suggestive of spasmophilia is still seen.

During 1942–1946, 43 children were selected for special observation. These children were under 2 years of age, well cared for, receiving vitamin D (prophylactically), mainly brought up on mother's milk, free from signs of rickets and with Chvostek's sign distinctly elicitable in both branches bilaterally with slight finger percussion. When first observed the average age of the children was 14.4 months. A follow-up study on an average of 4½ years after the first detection revealed persistence of the facial phenomenon in all the observed cases except 2.

The authors point out that this facial phenomenon present in older children may to a certain extent be elicited in the children in babyhood. Thus even in children under 2 years of age the Chvostek sign is of limited value for the diagnosis of spasmophilia. The etiology of this phenomenon is unknown. 8 references.

Transitory Renal Osteoporosis with Amino-Aciduria and Development of Allergy to Vitamin D (*Voorbijgaande renale osteoporose met amino-aciduric en ontwikkeling van een overgevoeligheid voor vitamine D*). *S. van Creveld and Ph. Arons, Amsterdam, The Netherlands.* Maandschr. v. kindergeneesk. 17 : 33–45, 1949.

This paper discusses hypophosphatemic renal rickets (Debré-de Toni-Fanconi syndrome), the features of which are bone changes resistant to vitamin D, low serum inorganic phosphorus, increased alkaline phosphatase, renal glycosuria, acidosis and aminoaciduria. A 17-month-old boy is described who had received 300,000 I.U. of vitamin D, orally three times during the first year of life, and who was hospitalized because of active rickets. There was a low serum phosphorus, increased alkaline phosphatase and low calcium. In the beginning he had latent tetany; with a later drop in serum calcium values the tetany became overt. There was slight glycosuria and aminoaciduria. Roentgenograms showed rickets. With 4,000 I.U. of vitamin D_2 orally no improvement was noted. Calcium gluconate with dihydrotachysterol orally (0.5 to 3 ml. daily) was followed by a small rise in serum calcium and some improvement in the x-ray picture. After one month 320,000 I.U. vitamin D_2 were given intramuscularly. Three months afterward the calcium level was low again, the phosphorus was still low and the alkaline phosphatase was still increased. A large dose of dihydrotachysterol effected rapid clinical and biochemical improvement and the child was discharged in good condition with instructions to take one dessert spoon of cod liver oil daily. After one month the serum calcium and phosphorus had again decreased and the alkaline phosphatase value had increased. Again dihydrotachysterol administration gave a favorable result. Vitamin D_2 was again given in a dose of 2,000 I.U. daily and again had an undesirable effect. The authors suggest that the Debré-de Toni-Fanconi syndrome developed as a result of the administration of three large doses of vitamin D during the first year of life,

accompanied by an allergic reaction to vitamin D. 16 references. 3 figures. 4 tables.—*Ph. Arons.*

The term Fanconi syndrome, also known as the Debré-de Toni-Fanconi disturbance, is applied somewhat loosely to a group of rare anomalous disturbances which have in common intractable low bicarbonate acidosis, renal glycosuria, vitamin D-refractory rickets, and low blood phosphate, with no retention of nitrogenous waste products in the blood. The comparative prominence of these separate disturbances varies from case to case. The urine, which is usually alkaline, contains an abundance of organic acids, including amino acids. An excessive amount of phosphate, calcium and ammonia is also present in the urine.

Less consistent phenomena shown by some of the cases have been physical retardation, bouts of dehydration and fever, hypoglycemia an hour or two after glucose ingestion, increased serum phosphatase, polyuria, proteinuria, or an acid urine.

McCune, Mason and Clarke postulated that glomerular function is normal in these patients (hence no azotemia), but that the ability of the renal tubular epithelium to reabsorb glucose, amino acids and phosphate from the glomerular filtrate is defective. From analysis of other cases on record they concluded that the Fanconi syndrome is related to hyperphosphatemic renal rickets on the one hand and generalized cystinosis on the other (Am. J. Dis. Child. 68: 81–146, 1943).

Lowering of the serum calcium, when present, is probably a consequence of the unusual output of urinary calcium. Albright and Reifenstein have suggested (The Parathyroid Glands and Metabolic Bone Disease, Williams & Wilkins, 1948) that the hypercalciuria stimulates a secondary hyperparathyroidism, which in turn evoke the hyperphosphaturia. The low serum phosphorus is a consequence of the hyperphosphaturia, whether the latter be due to parathyroid over-activity or to anomalous failure of the tubular epithelium to resorb phosphorus from the glomerular filtrate as McCune, Mason and Clarke have theorized.

With respect to the therapeutic management of the case reported here, it should be pointed out that both vitamin D and dihydrotachysterol influence calcium absorption from the intestine and phosphorus excretion into the urine. The much more satisfactory benefit secured with dihydrotachysterol should not be compared with the poor response to cod liver oil, for the reason that the oil was given in the ordinary prophylactic dose for a normal child of this age. Refractory rickets of this type must be treated with comparatively enormous doses, i. e., 50,000 to 300,000 I.U. daily. Parathyroid hormone would seem inappropriate, inasmuch as the parathyroids are already hyperactive. The suggestion that the condition developed as a sensitivity response to three large injections of vitamin D given in infancy is pure speculation, not substantiated in the other cases of this rare syndrome which have been recorded.—Editor.

26. Psychology, Psychiatry

Intelligence Quotient of Children Who Have Recovered From Erythroblastosis Fetalis. *Joan M. Gerver and Richard Day, New York, N. Y.* J. Pediat. 36: 342–48, Mar. 1950.

A survey was made of children who had recovered from erythroblastosis fetalis, and who were old enough so that a Stanford-Binet Intelligence Quotient could be obtained. No children were included who came to the attention of the authors because of suspected mental difficulty. Children for the study were found by searching neonatal records for cases of well-documented erythroblastosis fetalis. A total of 100 children were seen. Eleven of these had evidence of serious neurologic disease, thought to have been the result of kernicterus. In the group of 100 there were 68 children where the neonatal diagnosis was very satisfactory, where there was no neurologic difficulty apparent from physical examination and where there was an available older sibling who had not had neonatal disease. The mean I.Q. of these recovered children was 11.8 points lower than their unaffected siblings, a difference which was highly significant by proper statistical criteria. The inferiority in I.Q. was shown by inspection of the frequency distribution and by a 6 fold table not to be the result of the presence of a small group of seriously retarded children. There was likewise no correlation of I.Q. with age. In a group of 28 families free of erythroblastosis the same psychologist found no significant difference in the I.Q. of paired siblings with an almost identical age spread to the children from the families where there had been erythroblastosis. All the affected children were treated in the neonatal period with one or more small transfusions of blood. There was a low but statistically significant negative correlation between their subsequent I.Q. and the total amount of blood which had been given. There were no sex differences in I.Q. The depression in I.Q. is sufficiently slight so that there is no reason to alter the usually happy prognosis given in cases where recovery from erythroblastosis has occurred without neurologic sequelae. 5 references. 1 figure. 3 tables.—*Author's abstract.*

This is an interesting study but caution must be observed in accepting older siblings as controls in studying the I.Q. The differences noted here may be significant statistically but physicians generally will not be much disturbed by a difference between an I.Q. of 103 and one of 115.—Editor.

Twelve Months' Study of Glutamic Acid Therapy in Different Clinical Types in an Institution for the Mentally Deficient. *Karl V. Quinn and Dorothy Durling, Wrentham State School, Wrentham, Mass.* Am. J. Ment. Deficiency 54: 321–32, Jan. 1950.

Thirty-one children of various clinical types were treated with glutamic acid for 6 months. The 25 patients who were tested with the

Revised Stanford-Binet Intelligence Test, Form L, gained on the average of 3.9 I.Q. points. The glutamic acid powder was administered in 12 Gm. amounts with the morning cereal, in one daily dose. This was well taken and did not provoke any gastric or physical distress. Eighteen additional children, in 11 of whom the diagnosis was postinfectional, were treated with glutamic acid plus vitamins for 6 months. The 12 patients tested with the Revised Stanford-Binet Intelligence Test, Form L, gained on the average 3.3 I.Q. points. Fourteen of these were continued for a second 6 months' course of glutamic acid treatment. For the 13 children tested with the Revised Stanford-Binet Test, Form L, the average gain for the first 6 months of treatment was 4.8 I.Q. points. The I.Q. gain for the full year was 4.4 I.Q. points.

It was concluded that glutamic acid appears to stimulate mental and physical activity in some mentally deficient children. There may be a gain in I.Q. of 5 to 10 points, at least temporarily. The most substantial part of this improvement takes place during the initial 6 months of treatment. Relatives feel that something material has been added to their defective child's advancement. 7 references.

The Treatment of Mentally Retarded Children with Glutamic Acid (*De behandeling van geestelijk achtergebleven kinderen met glutaminezuur*). *W. Ernsting, Hoorn, The Netherlands.* Nederl. tijdschr. v. geneesk. 93: 1044–54, April 2, 1949.

Eight of 12 mentally retarded children were given l-glutamic acid in increasing dosage up to 10 Gm. per day over a period of 6 months. Four other children were used as controls and received lactose. Treatment had to be discontinued on 1 patient owing to abdominal complaints; 1 other patient had a transient itching exanthem on the back. The children were tested with the Binet-Simon (Stanford revision), Pinter-Cunningham and Pinter-Peterson tests. The average advance in mental age was 4½ months. The I.Q. did not increase in any case. In 2 children it remained constant, in 6 it was lowered. The control group had slightly better results. The I.Q. of 2 children became lower, it remained stationary in 1 and was improved in 1, perhaps because of changed exterior circumstances. In assessing an apparent improvement in intellectual capacity, increasing familiarity with the tests must be taken into account. It is essential that no environmental changes occur. 17 references. 2 tables.—*M. G. Stronk.*

As demonstrated by the above three abstracts, there is no unanimity as to whether or not glutamic acid helps mentally retarded children. Certainly the benefit it gives, if any, is not striking.—EDITOR.

27. Public Health, Epidemiology

1948 Infant Mortality Rate for the United States the Lowest on Record. *News Release.* U. S. Public Health Service, Federal Security Agency, Washington, D. C.

The number of deaths under 1 year of age reported in the United

States during 1948 was 113,169. The number of births was 3,535,068, giving a rate of 32.0 deaths per 1,000 live births. The rate had been 32.2 in 1947 and 47.0 in 1940. Provisional figures indicate a further decline in 1949 to an estimated rate of 31 per 1,000 live births. By states, the infant mortality rates in 1948 ranged from 24.3 for Connecticut and 25.5 for Oregon to 46.2 for Texas, 56.4 for Arizona, and 70.1 for New Mexico.

The mortality risk proves to be greater in the first day of life than at any other period during infancy. One third of the deaths in the first year of life occur in the first day, a second third (36.0%) in the remainder of the first month and the rest (30.7%) at ages between 1 month and 1 year. In every one of these age intervals (Table 2)

Table 2—*Deaths Under One Year and Infant Mortality Rates by Age, Race, and Sex: United States, 1948*
(Exclusive of stillbirths. Rates per 1,000 live births in each specified group)

Age	Race			Sex	
	Total	White	Nonwhite	Male	Female
Under 1 year	113,169	92,034	21,135	65,151	48,018
Under 1 day	37,652	31,768	5,884	21,977	15,675
Under 1 month[1]	78,526	65,182	13,244	45,770	32,656
1– 5 months	24,282	18,685	5,597	13,762	10,520
6–11 months	10,461	8,167	2,294	5,619	4,842
	RATE				
Under 1 year	32.0	29.9	46.5	35.9	27.9
Under 1 day	10.7	10.3	12.9	12.1	9.1
Under 1 month[1]	22.2	21.2	29.1	25.2	19.0
1– 5 months	6.9	6.1	12.3	7.6	6.1
6–11 months	3.0	2.7	5.0	3.1	2.8

[1] Includes deaths under 1 day.

Table 4—*Infant Mortality Rates for Specified Causes: United States 1930, 1940 and 1948*
(Exclusive of stillbirths. Rates per 1,000 live births)

CAUSE OF DEATH	1948	1940	1930[1]
ALL CAUSES	32.0	47.0	64.6
Whooping cough	0.2	0.9	1.5
Tuberculosis (all forms)	0.1	0.2	0.5
Dysentery	0.2	0.4	0.4
Syphilis	0.1	0.5	0.8
Diarrhea, enteritis, and ulceration of the intestines	1.8	3.5	7.8
Pneumonia (all forms) and influenza	3.7	7.4	10.2
Congenital malformations	4.5	4.7	5.3
Congenital debility	0.5	1.2	2.5
Premature birth	11.1	13.7	16.7
Injury at birth	3.4	4.5	4.8
Asphyxia (cause not specified) atelectasis	1.5	1.3	1.0
Accidental deaths	1.0	1.1	1.0

[1] In 1930, Texas and South Dakota were not included in the birth registration area.

the infant mortality rates for males are higher than those for females, and the rates for nonwhite infants higher than those for white infants.

The infant mortality rate has been reduced by 50% since 1930, when it was 64.6 deaths per 1,000 live births. The greatest gain has been in the rate for infants 6 to 11 months of age. In this group, the rate has declined from 10.7 in 1930, to 3.0 in 1948. A smaller reduction in mortality took place for infants 1 to 5 months of age (62%) and an even smaller decline (38%) in the first month of life. With the greater declines in the death rates for the contagious and infectious diseases, the causes associated with conditions before and at birth have become more prominent (Table 4). 4 tables.

Infant Mortality in Spain for the Period 1941–1944 (*La mortalidad de la infancia en Espana, cuatrienio 1941–1944*). *A. A. Curbelo, State Child Health Office, Madrid, Spain.* Rev. espan. pediat. 6: 190–215, March–April 1950.

There were 30.42 neonatal deaths (under 1 month of age) per 1,000 live births in Spain from 1941 to 1944. Congenital disease, including prematurity, accounted for 62.66%. Respiratory infections were second in importance: 13.80%. Ten per cent were due to enteric infections. 35 references. 8 tables. 5 graphs.—*A. M. Bongiovanni.*

28. Respiratory System

Bronchopulmonary Mycosis. *Brenton M. Hamil, Detroit, Mich.* Am. J. Dis. Child. 79: 233–71, Feb. 1950.

The unusual simultaneous primary occurrence of bronchopulmonary mycosis (Aspergillus niger and Monilia pinoyi) in 5 members of a family group is presented. The literature relating to the history of developing interest in the disease and modern concepts of diagnosis and treatment are reviewed. A similar instance has not been reported.

The course of the disease was observed over a period of ten years. Serial blood counts and sedimentation rates are presented in correlation with types of treatment instituted and the interpretation of roentgenograms of the chest during progress of healing.

Presentation of reproductions of roentgenograms for each patient shows the changes from soft, fluffy parenchymal densities from apices to bases in the early stage of the disease through a state of apparent condensation of these to a fibrotic appearance, and later the deposition of calcium in a few, then all of the nodules as healing progressed, with final condensation of these calcium densities scattered throughout the lungs. The earliest appearance of calcium in the nodules was about three years after intermittent treatment, and within a few months after intensive treatment with iodides, mapharsen and autogenous vaccines.

The results of serological tests and of skin test reactions to serial in-

creasing dosages of various tuberculin materials and to autogenous and stock vaccines of various fungi and of the bacterial organisms common to the upper respiratory tract, at intervals during progress of healing, are presented. The extent of allergic response to heterologous fungi but more specific response to autogenous organisms is evident.

Successful treatment depends upon early diagnosis and rests chiefly upon administration of adequate amounts of iodides. Potassium iodide can be given safely in massive doses by mouth for long periods without toxic effects. Sodium iodide can be given as 10% solution intravenously, daily, for long periods simultaneously with potassium iodide. Ethyl iodide can be given effectively by inhalation for short periods without known toxic or other unfavorable effects other than those common to iodides in general. Blood counts and sedimentation rates are helpful in evaluating the progress of the patient and the activity of the disease. These data in tuberculosis are similar, nullifying their value in differential diagnosis. Primary bronchopulmonary mycotic infection with Monilia pinoyi and Aspergillus niger healed completely in the 4 children observed, leaving scattered dense miliary calcium nodules throughout the lungs. There were no residual symptoms other than spastic paralysis in 1 patient in whom transverse myelitis developed during the course of the disease. 56 references. 8 figures. 5 tables.—*Author's abstract.*

29. Skin, Teeth, Hair

See Contents for Related Articles

30. Social, Economic and Organizational Problems

CONSTITUTION OF INTERNATIONAL PEDIATRIC ASSOCIATION

(Adopted in Zürich, Switzerland, July 26, 1950.)

Article I—Name

The name of this association shall be "The International Pediatric Association."

Article II—Objects

The objects of this association shall be:

1. to promote closer relationships between the pediatricians of all countries by holding an international congress every 3 years.

2. to promote the dissemination of pediatric knowledge.

3. to cooperate with other agencies concerned with the improvement of child care, pediatric education or research in any part of the world.

Article III—Membership

Membership shall be composed of national pediatric associations. These associations shall pay dues in proportion to their per capita

memberships, as determined by the International Pediatric Association. Applications for membership shall be made to the general secretary of the association. Membership shall be terminated by resignation or nonpayment of dues.

Members of member associations shall automatically be members of the International Pediatric Association. It shall be the obligation of the secretary of each member-association to furnish the general secretary of the International Pediatric Association with a list of the officers and members of his national association on January 1st of each year.

Members shall have the privilege of submitting communications or exhibits to the Congress. The program committee of the Congress may, however, exercise its discretion in selecting these and may solicit communications or exhibits from any source.

Article IV—Governing Bodies and Officers

A. General Council

The control of policies of the International Pediatric Association shall be vested in a General Council, consisting of one delegate from each member-association. Each delegate shall have one vote, except that in the case of a country having more than one member-association only one vote shall be accorded that country.

The General Council shall meet every 3 years at the time of the Congress.

B. Officers

The officers of the Association shall be: a permanent General Secretary who shall also act as *Treasurer*, the President of the Congress and an Executive Board of 8 members. The General Secretary-Treasurer and the President of the Congress shall be ex officio members of the Executive Board, the remaining 6 being members-at-large. The term of office shall be from the termination of one Congress to the termination of the next. The Officers of the Association shall be elected by the Council. Three of the six members-at-large of the Executive Board shall be eligible for reelection to succeed themselves for a second term.

The Executive Board shall elect its own chairman. Its functions shall be to approve applications for membership in the Association and to carry out the policies of the Association between meetings of the Council.

Article V—Congress

The General Council shall determine the location of the triennial Congress, the exact time to be determined by the president in consultation with the Council for the Coordination of International Congresses of Medical Sciences. The official languages of each congress shall be determined by the president, who shall have the authority to appoint

such committees as are necessary for the conduct of the Congress which shall be sponsored by the national member-association or associations of the country in which it is held.

By-Laws of the International Pediatric Association

1. Dues. Dues for member-associations shall be recommended by the executive board and approved by the general council. They shall be based on the per capita membership of each member-association. They shall be payable in advance on January 1st of each year. Non-payment of dues for 2 years will automatically terminate membership, due notice being given. It may also be terminated by vote of the council.

2. Voting. A two-thirds vote of those present at the meeting of the General Council shall be valid for changing the constitution. A majority vote of either body shall be valid for all other matters.

3. Congress Attendance. Attendance at the Congress shall be open to any reputable physician on payment of registration fee. Only by special invitation, however, can physicians who are not members of a member-association participate in the discussions.

Professor Fanconi of Zurich has been elected Secretary-General and Treasurer of the International Pediatric Association. The Executive Council for the next 3 years consists of Drs. Helmholz, Gomez, Holt, Wallgren, Moncrieff, Debré, Maslov and, ex officio, Hurtado and Fanconi. The annual dues for member-associations are 1 franc (Swiss) per member for each member-association.

The next International Congress of Pediatrics, which is the seventh, will be held in 1953 in Havana, Cuba. Professor Felix Hurtado of Havana will serve as president.

31. Surgery, Anesthesia

Anesthesia in Infants and Young Children for Major Surgical Procedures. *C. R. Stephen, Montreal, Canada.* Arch. Surg. 60: 1035–44, June 1950.

In considering narcosis for the young, the anesthetist must bear in mind that the margin of safety is much narrower than that found in adults. At all times he must be careful in detail, meticulous in observation, gentle in maneuver and skillful in technic. Premedication with morphine and scopolamine or atropine is valuable in lessening apprehension and in promoting a smoother, less stormy induction.

Any anesthetic agent which has been used safely in adults can be employed in children. Ethyl ether is still considered the safest agent known. Nitrous oxide disturbs metabolism little and has a high analgesic value. When it is employed, with never less than 25% oxygen as a vehicle for the volatilization of ether, the amount of the latter agent can be reduced considerably.

It should be understood that the technic used to produce narcosis is of greater importance than the agent employed. Any technic devised for anesthetizing small children should have the following aims in view: abolition of dead space; reduction of resistance to a minimum; prevention of accumulation of carbon dioxide; and maintenance of as even and light a plane of anesthesia as is compatible with the surgical procedure. It has been found over a trial-and-error period, during which different technics were evaluated, that one which employs a nonrebreathing valve close to an endotracheal tube tends to fulfil best the above objectives. Examples are given to demonstrate the utilization of this and other technics in major surgical procedures in infants and children. 5 references. 5 figures. 4 tables.—*Author's Abstract.*

32. Tumors

Carcinoma of the Cervix in the First Year of Life. Report of Two Cases. *George P. Heckel, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.* Pediatrics 5: 924–29, June 1950.

Two girls, 7 and 11 months of age, with adenocarcinoma of the cervix, are described. There is probably one previously reported case occurring under 1 year of age. The younger infant died of the disease on the day of admission to the hospital, having had vaginal bleeding for about a month. Autopsy showed adenocarcinoma of the cervix uteri extending to vagina, fundus uteri, fallopian tubes and ovaries, with metastasis to the retroperitoneal lymph nodes; hydroureters and hydronephrosis; bilateral cystic ovaries; and anemia. The second child had vaginal bleeding which began eight days before operation. Insertion of a small Kelly cytoscope into the vagina revealed friable redundant tissue in the vault from which a piece adequate for histologic study adhered to the instrument. It proved to be adenocarcinoma. She is surviving normally 21 months after total hysterectomy. 9 references. 4 figures.

Teratoma of the Ovary in an Infant 20 Months of Age. *Sherman S. Garrett, Champaign, Ill.* Am. J. Dis. Child. 79: 321–23, Feb. 1950.

Teratoma of the ovary is a tumor rarely found in young children. This case appears to be the earliest so far described. The patient, delivered by cesarean section, was the first born to a mother aged 28. The child had always appeared normal and there were no complaints. On routine examination at the age of 20 months, a smooth ovoid mass the size of a large egg was noted in the suprapubic region, in the midline, with the long axis vertical. The tumor was freely movable and could be manipulated into any part of the abdomen without discomfort to the patient.

The tumor, which had grown slowly, was removed at operation 7 months later through a mid-left rectus incision using ether anesthesia

(age, 27 months). The mass apparently originated in the left ovary. Recovery was prompt and uneventful. On histologic study it proved to be a papillary serous cyst of the ovary; teratoma of the ovary.

Harris (1917) was able to collect reports of 21 cases of teratoma of the ovary in patients under 14 years. The youngest of these was aged 3 years. Mazzola and Ryan described a dermoid at age 5; Ladd and Gross noted one at age 6. German reported a teratoma of the ovary in a patient at 6 years and 10 months. Ovarian tumors in young children should be removed promptly to avoid the possibility of allowing malignant changes to develop in a benign growth. 5 references. 1 figure.

The Pediatric Bookshelf

Tuberculosis and Other Problems in Pediatrics. *Arvid Wallgren, M.D.* Baltimore, Williams & Wilkins Co., 1950. $2.25.

Arvid Wallgren, internationally known pediatrician and authority on childhood tuberculosis, delivered the tenth series of the Abraham Flexner lectures at Vanderbilt University. These five lectures have been collected in book form. Three lectures deal with tuberculosis, the remaining two with problems of illegitimacy and erythema nodosum.

In Sweden, tuberculosis in childhood has been reduced almost to the vanishing point by a program of prevention and treatment, which is popular with the public and strongly supported by law. In tuberculin-negative children the incidence of tuberculous infection is reduced by the BCG vaccination, the early findings and isolation of persons with positive sputum, and the elimination of tuberculous cattle. Once a child has become tuberculin-positive by natural infection, efforts are limited to strengthening natural resistance and general health. Dr. Wallgren does not believe that continued exposure to tuberculosis has any bad effect on tuberculin-positive children. Treatment is generally of a nonspecific type. Children are not hospitalized unless adequate care cannot be given at home. Streptomycin is used in infancy with the hope of preventing hematogenous dissemination.

In Sweden, BCG vaccination of tuberculin-negative persons is almost universal. The production of general tuberculin sensitivity in the population has not seriously increased the difficulty of diagnosing the disease. Since the incidence of tuberculosis is low, natural infection does not occur usually until after the childhood years of greatest resistance. It is, therefore, wise to produce some immunity with an artificial nonvirulent infection before adolescence. For practical purposes this immunity is present as long as a vaccinated person continues to react to tuberculin. BCG does not provide absolute protection against infection with virulent acid-fast organisms. It protects against virulent primary and early postprimary infections. The effect on the development of osseous and renal and late pulmonary disease is less marked.

Discussing erythema nodosum, Dr. Wallgren feels that this is a nonspecific allergic cutaneous eruption. The great majority of cases in Sweden are associated with tuberculosis and occur at the time of the conversion of the tuberculin from negative to positive. The occurrence of the eruption does not affect the prognosis of the case.

One out of ten children in Sweden is illegitimate. The nation faces this problem realistically by providing care for the mother before and after delivery of the infant and by establishing a legal guardian for the child. The success of these measures is reflected in the sharp decrease in the disproportionately high death rate of these children.

Dr. Wallgren makes the diagnosis of pyloric stenosis by demonstrating, on x-ray examination, a narrow canal of barium in the pyloris. Most such children are treated without surgery. Their symptoms are relieved but the roentgenological findings persist. Although some of the opinions expressed in this book are contrary to common practice in this country, Dr. Wallgren's vast experience makes it advisable to become acquainted with his point of view.—*E. Sewell.*

Clinical Pathology. Application and Interpretation. *Benjamin B. Wells, M.D., Ph.D., Professor of Medicine, University of Arkansas School of Medicine, Little Rock, Arkansas.* Philadelphia, W. B. Saunders Co., 1950. $6.00.

Here is an excellent survey of the "in-between" zone of knowledge in which the results of laboratory tests become correlated and interpreted with the findings exhibited by patients. Diagnosis and treatment and the evaluation of nutritional status can be helped immensely by the intelligent use of simple laboratory aids. This book is designed primarily for the internist, but most of the contents are equally applicable to problems of the older child and adolescent.

Normal Values in Clinical Medicine. *F. William Sunderman, M.D., Ph.D. and Frederick Boerner, V.M.D.* Philadelphia, W. B. Saunders Company, 1949. $14.00.

Assisted by a large group of collaborators, the authors have gathered from published reports a series of 74 chapters of so-called "normal" data. The fields of electrocardiology, hematology, blood and lymph chemistry, respiratory system, nervous system, urinary tract, muscles, skin, endocrines, metabolism, teeth, food values and sundry other aspects of the human organism are all considered. There are 237 illustrations and 413 tables, each accompanied by a proper bibliographic reference. Most of the text refers to adults, but considerable attention is also given to the phenomena of childhood. The pediatric field has not been fully surveyed—summaries of such classic studies as those of Macy, Todd, Washburn and Stuart, for example, have not been reproduced. Several series of height-weight tables are given—those of Barach (1945), Crum (1916), and Prior and Stolz (1933).

One defect of this volume (for which the desire to avoid over-bulkiness is no doubt partly responsible) lies in the absence of statements with respect to the character of the original population from which the reproduced charts and tables were derived. The research investigator will undoubtedly want to refer back to the original sources to make sure that the data cited for his field of interest are fully representative and adequate. The practitioner who wants a ready encyclopedic-like collection of figures for aid in the clinical evaluation of problem patients, on the other hand, can conserve a great deal of time by keeping this volume readily available.

The Rh Blood Groups and Their Clinical Effects. *P. L. Mollison, A. E. Mourant and R. R. Race.* Medical Research Council Memorandum No. 19. London, His Majesty's Stationery Office, 1948. 74 pp. 1s. 6d.

This concise exposition is divided into three principal sections. The section on Classification, by Race, presents the British nomenclature and the Fisher-Race theory of inheritance. The section on Clinical Considerations, by Mollison, is designed for the pediatrician, obstetrician and general practitioner. The section on Rh Testing, by Mourant, describes methods of importance to the clinical pathologist. This is an excellent introduction to the newest subspecialty of hematology.

Proceedings of the First Clinical ACTH Conference. *Edited by John R. Mote, M.D., Medical Director, Armour Laboratories, Chicago.* Philadelphia, The Blakiston Co., 1950. $5.50.

This is a compilation of the papers read at the first conference on ACTH, held at the Hotel Stevens, Chicago, on October 21 and 22, 1949. It consists of 52 separate reports on clinical investigations into the metabolic, physiologic and therapeutic effects of the pituitary adrenocorticotropic hormone. The reports are all provisional, and over the next one or two years should appear in expanded form scattered through the body of medical literature. Many of the papers and the appended discussions deal with problems related to childhood. Every physician who plans to use ACTH, now that it is generally available, should first familiarize himself with the trends of its multifarous primary and secondary actions as collected in this volume.

Epidemics in Schools: An Analysis of the Data Collected During the Years 1935 to 1939; British Medical Research Council Special Report Series No. 271. *E. A. Cheeseman.* London, His Majesty's Stationery Office, 1950. 75 cents. American Agents: British Information Services, 30 Rockefeller Plaza, New York, N. Y.

An earlier special report, No. 227, on epidemics in schools has proved a valuable reference for those interested in infectious disease and epi-

demiology. This new special report, No. 271, as indicated in the preface, supplies additional related data. The two together give a reliable and clear picture of the nature and amount of sickness experienced by boy and girl pupils in boarding schools. Painstaking and statistically reliable analyses of this sort provide information on the natural history of common epidemic diseases not available elsewhere. The demonstration that such diseases as chickenpox, usually considered highly infectious, may be limited to one case in an outbreak, is a sobering thought for those committed to studying methods of prophylaxis and epidemic control. Dr. Cheeseman is to be congratulated on getting these data together despite the dislocation of personnel and records occasioned by the War, and the Medical Research Council on its vision to see the need for such information and arrange for its collection.

The Psychoanalytic Study of the Child, Volume III and IV, A Collection of Essays by Different Authors. New York, International University's Press, 1949. $10.00.

This is a companion to similar studies by a distinguished group of Freudian psychoanalysts which have appeared under similar auspices and were reviewed in this Journal. Several of the papers encompass material which interests the pediatrician in his everyday practice. These essays will be particularly readable by those physicians having had either a personal psychoanalysis or having become familiar with Freudian theory and practice through some other media. Even so the book does not make for light or casual reading.

Of particular interest to the pediatrician will be an essay from investigators at the Menninger Clinic. The subject discussed is titled "The Reaction of Infants to Stress." The authors undertook a systematic and painstaking observation of what infants do when exposed to various stimuli which are supposed to evoke tension. The modes of expression are described in detail, often through the use of case histories. Clinical knowledge of the pediatrician was corroborated in this laboratory study when it was discovered that in general, tension is reflected in disturbances in physiological functioning more readily by infants than by adults and that these upsets are particularly frequent in the gastrointestinal tract, the skin, the respiratory tract, and the temperature control mechanisms.

Although this book covers several areas, including material on aggression, the relation of twins to each other, screen memories, dreams of children of the preschool age, a psychoanalytic evaluation of ties and delinquency, three of the chapters should interest the pediatrician particularly. One of these deals with the physical symptoms seen in emotionally disturbed children, the second discusses neurosis and home background, and the third is a survey of the literature pointing out the relationship of feeding problems to psychogenic factors.

Like its predecessors, these Volumes III and IV of the Psychoan-

alytic Study of the Child make important contributions to the theory and clinical observations of child behavior. It is unfortunate that they will have greatest usefulness to those relatively few pediatricians who have good understanding of Freudian psychoanalysis.—*Milton J. E. Senn.*

The Handicapped Child. *Edith M. Stern with Elsa Castendyck.* New York, A. A. Wyn., Inc., 1950. 179 pp. $2.00.

Parents whose children are handicapped either in their senses or in their limbs are often denied the joy of seeing their child growing up normally, and are plagued by both fears and guilt. As Mrs. Stern and Miss Castendyck point out, this is neither a general manual on child care nor an encyclopedia in terms of illnesses. It stresses "the fact that good principles of child rearing apply equally to the normal and the handicapped."

With an extremely frank survey of the feelings of the parents and an appreciation of what the handicapped child means to them, the authors have tried to relieve the unnecessary guilt of parents who frequently feel themselves at fault. They point out the effects of this guilt, either in smothering the child with attention, or in being overly demanding. They point out how human and ordinary it is to become "fed-up" or overwhelmed with the situation of having a handicapped child. They point out the routes that parents can take to get help both for themselves and for the child.

Following this general statement the book is divided by conditions, covering crippled children with cerebral palsy, epilepsy, and those with impaired sight, vision, and mentality. There is a final chapter on chronic illness, particularly rheumatic fever. Each of these chapters provides concrete advice about the particular condition and offers valuable suggestions as to where the parents may turn. The book tries to make clear the crippling aspects of the handicapped, both physically and emotionally, and to help parents clarify their feelings about the child as they work to help him.

While it is obviously impossible to solve all the problems faced by parents whose children are different from others, this book, stressing as it does the underlying principles of good child care, can be of real assistance and support.—*Henry H. Work.*

The Adrenal Cortex. *Consulting Editor: Robert Gaunt.* Vol. 50, Art. 6, pp. 509–678. Annals of the New York Academy of Sciences.

This is a comprehensive symposium on the biochemistry and physiology of the steroids coming from the adrenal cortex. There are several excellent summaries of clinical investigations, including one on Stress Responses by Gregory Pincus, on Addison's Disease by George W. Thorn and Associates, and on Cushing's Disease by Edwin J. Kepler.

QUARTERLY REVIEW

of

PEDIATRICS

VOLUME 5

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The "when" and "why" of calcium therapy, therefore, are clear and the question of greatest concern becomes: *What is the best source of this mineral element?* Milk is an excellent source but is not always practical; many patients cannot or will not take adequate milk. Moreover, dietary elements such as fats and vegetable oxalates interfere with calcium absorption from foodstuffs.

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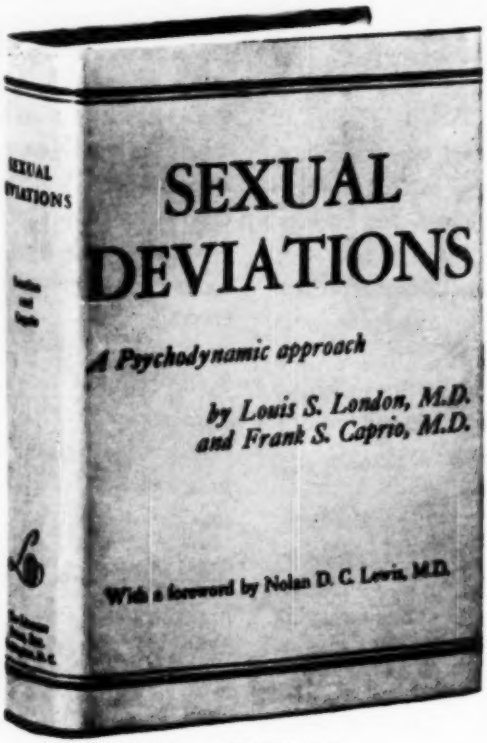
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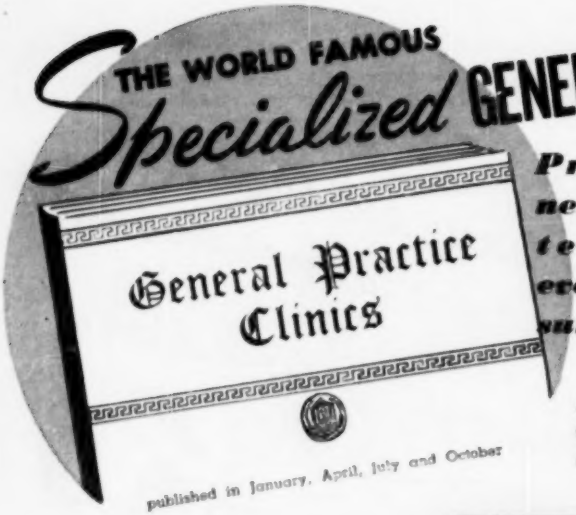

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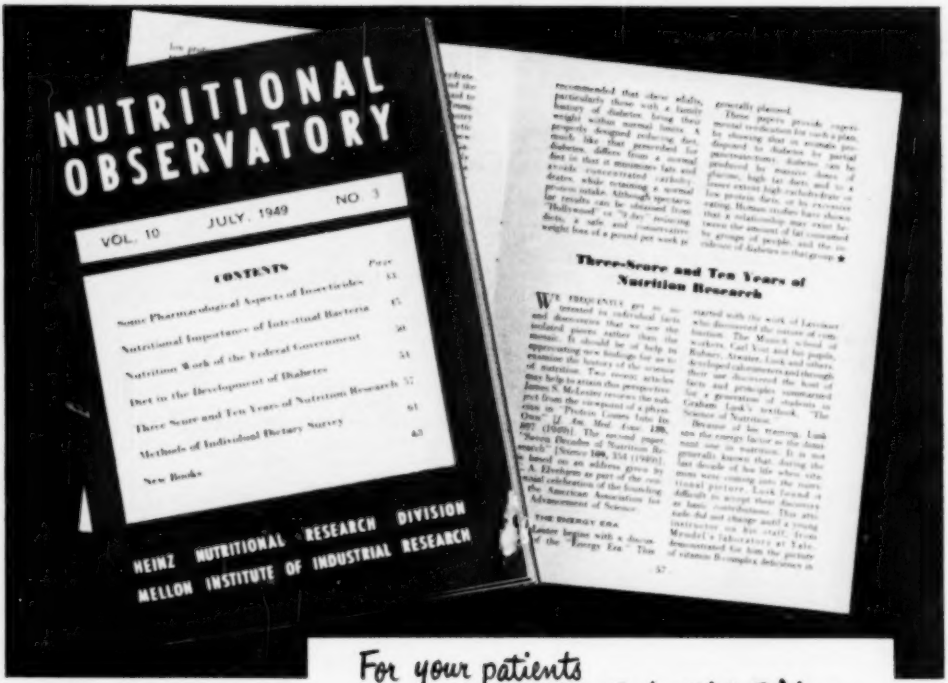
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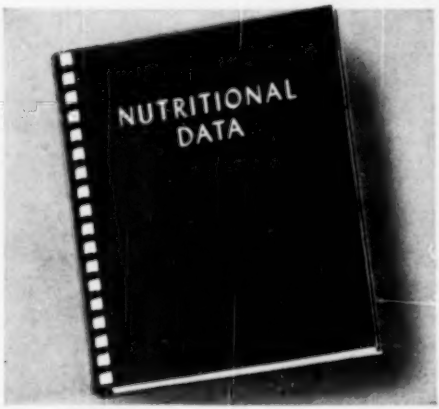

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1. Follis, R. H., Jackson, D., Eliot, M. M., and Park, E. A.: *Am. Jour. Dis. Child.*, 66:1, July, 1943.
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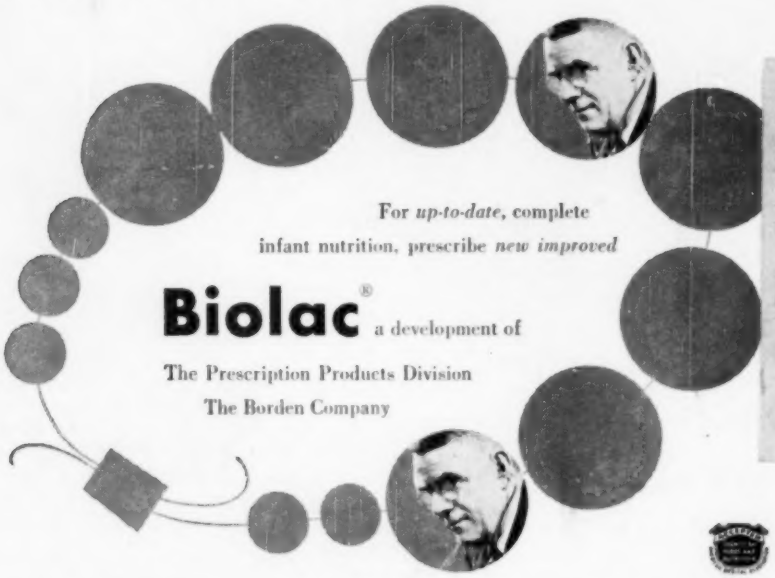

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Ann Arbor, Michigan

Ingredients: skim milk, dextrins-maltose-dextrose, lactose, coconut oil, destearinated beef fat, lecithin, sodium alginate, disodium phosphate, ferric citrate, vitamin B_1, concentrate of vitamins A and D from fish liver oils, and water. Homogenized and sterilized.

Dilution: one fluid ounce to one and a half ounces of boiled water for each pound of body weight.

Biolac is available at drugstores in 13 fluid ounce tins.

The Borden Company, Prescription Products Division, 350 Madison Avenue, New York 17